

# Annals of Otology, Rhinology and Laryngology

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THE INDEX OF OTOLARYNGOLOGY

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LIX.

THE MECHANISM OF PAIN TRANSMISSION IN  
CERTAIN TYPES OF OTALGIA.\*

BY RALPH A. FENTON, M. D.,

AND

OLOF LARSELL, PH. D.,

PORTLAND.

Transmission of pain impulses from the complex sphenopalatine region of the nose has for many years engaged the attention of clinicians in otolaryngology. With the advent of cocain, and later of adrenalin, relief of neuralgias in the ear, mastoid, occiput, trapezius, tongue, pharynx and larynx has been ascribed to the action of these drugs upon the sphenopalatine ganglion.<sup>1</sup> Transmission of pain impulses through sympathetic fibers has been imagined by certain clinicians. An entire system of therapeutics, including destruction of the sphenopalatine ganglion by alcohol or by surgical attack, ablation of the middle turbinate, opening of the apparently uninvolved sphenoid has been devised, with very considerable

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\*Delivered before the First International Oto-Rhino-Laryngological Congress, Copenhagen, July 29-August 1, 1928.

success in many cases and absolute failure in many others. Treatment of associated myalgias of the neck has been disappointing, and their pathogenesis remains obscure.<sup>2 3 4 5</sup>

Inadequate knowledge of the histopathology and neurologic connections of this region is confessed not only by Sluder,<sup>6</sup> its most courageous invader, but also by such painstaking investigators as Müller<sup>7</sup> and his collaborators; by Bertein,<sup>8</sup> Terracol<sup>9</sup> and Halphen<sup>10</sup>; by Lasagna<sup>11</sup> and by Segura.<sup>12</sup> In Halphen's words, "nous avançons dans un formidable inconnu . . . notre pathogénie nous paraît-elle bâtie sur des sables bien mouvants."

Investigation of the neurohistology of this region, because of difficulty of securing absolutely fresh material, and of the inadequacy of degeneration studies of fibers and cells in an area so complex, has been carried on mainly by comparative anatomists. According to Büscher, "Das Studium der sympathischen Nervenfasern wird dadurch erschwert, dass diese sich aus *marklosen*, aus *dünnen* markhaltigen Nervenfasern und vereinzelt aus solchen mit *breiter Markscheide* zusammensetzen . . . . An den dünnen Markfasern ist eine degeneration recht schwierig zu erkennen; fast unmöglich ist dies an den marklosen . . . Fasern."<sup>13</sup> Van Gehuchten<sup>14</sup> produced degeneration of cells in the geniculate ganglion of the rabbit by section of the seventh nerve at its exit from the facial canal. Yagita<sup>15</sup> showed chromatolysis of many cells in the geniculate ganglion following section of the great superficial petrosal. Following clinical study by Ramsay Hunt<sup>16 17 18</sup> of herpes oticus (in which he maintained the existence of sensory fibers in the seventh nerve), Gordon Wilson was unable to achieve accurate degenerative studies in the higher apes by destructive lesions of the geniculate ganglion itself.<sup>19</sup> Müller and his collaborators remain uncertain as to the function of the deep petrosal,<sup>7</sup> and whether sensory pathways exist through the cervical sympathetic.<sup>20</sup> Bürger,<sup>21</sup> in his recent masterly review, points out the existing confusion between sympathetic phenomena and true sensory manifestations from the sphenopalatine region.

Recognizing these defects in the explanations of referred pain from the sphenopalatine area, we have recently made a

study of its comparative anatomy and embryology, and have examined human and mammalian material by serial sections of the sphenopalatine ganglion and connected nerves, in an effort to establish accurate anatomic pathways. Part of this work was recently presented before the American Otological Society<sup>22</sup> and forms a foundation for our present conclusions.

The anatomic connections of the sphenopalatine ganglion suggest interesting possibilities. We should keep clearly in mind that its central position in an intricate plexus of nerve trunks, autonomic and sensory, is not alone the important factor. (Fig. 1.) Rather, the morphologic significance of some of the nerve trunks entering the ganglion is of special interest. The principal connection of Meckel's ganglion is the Vidian nerve, made up of the great superficial petrosal and the great deep petrosal nerves. The former is homologous, in part, with the palatine branch of the facialis of lower forms, as will be shown below. The great deep petrosal nerve is, in the human, made up of unmyelinated postganglionic sympathetic fibers from the superior cervical ganglion; but it contains a considerable number of large and medium sized myelinated fibers which appear to be derived from other sources, perhaps from the petrous ganglion of the glossopharyngeus, and which probably reach the great deep petrosal nerve by way of the nerve of Jacobson and the small deep petrosal nerve. Such connections would make the sensory fibers, if such they be, of the great deep petrosal, homologous with the palatine branch of the glossopharyngeus as found in fishes. The connecting rami between the petrous ganglion and the nodose ganglion of the vagus offer further opportunities of communication, but all that can be stated of them at present is that in the rabbit they contain many large myelinated fibers, as shown by osmic acid staining. Much more work, now in progress, is necessary before any adequate statement can be made of the neural relations of the great deep petrosal, on the one hand, to the glossopharyngeus and vagus, and possibly to the spinal accessory, and, on the other hand, of these nerves, particularly the latter, to pain in the neck (sternomastoid) and shoulder (trapezius) regions.

Concerning the relations of the great superficial petrosal nerve to the sphenopalatine ganglion and to the facialis nerve, we can now present more complete findings. (Fig. 2.) The sphenopalatine ganglion is embryologically formed by migration of cells from the fifth and seventh ganglia,<sup>23</sup> and may also retain connections with the glossopharyngeus and vagus as just mentioned.

Various functional components exist in the cranial nerves of the lower vertebrates which are lost or greatly modified in man. Five distinct physiologic sets of nerve fibers may be identified within the seventh and ninth nerves and their branches, and these have been so carefully studied by comparative anatomists and morphologists that they may readily be traced. Their central connections are accurately identified within the brain, and their course in the nerve trunks has been followed by taking account of histologic variations, notably of size of fibers, difference in diameter and thickness, or absence of the myelin sheath, etc.

The complex process of cephalogenesis has led in higher forms to a grouping of similar components together within the brain stem. Thus, the fasciculus solitarius contains the visceral afferent fibers of the seventh, ninth and tenth nerves, while all the general cutaneous fibers enter the spinal fifth tract and nucleus. Similarly, the fifth nerve in mammals has peripherally mostly sensory, and the seventh nerve mostly motor components; this although in the lower vertebrates both nerves have mixed components, very much alike. Similar comparisons may be made for other cranial nerves.

No sphenopalatine ganglion is present in the sharks (Fig. 3), but rostrally directed palatine branches proceed from both the facialis and the glossopharyngeal. The ramus palatinus of the seventh derives from the geniculate ganglion. In the amniotes (Fig. 4) the sphenopalatine ganglion first appears along the palatine seventh, while the palatine ninth has disappeared. However, the ninth and seventh remain connected through the nerve of Jacobson, and there is also a branch between the nodose ganglion of the tenth and the petrous ganglion of the ninth.<sup>32</sup>

Following the views of Retzius,<sup>33</sup> Müller and Dahl,<sup>34</sup> and Carpenter,<sup>35</sup> we have confirmed their observations of the sympathetic character of the sphenopalatine ganglion, demonstrating therein numerous typical multipolar cells encircled by fine fibrillary end nets. (Fig. 5.) The fibers which give rise to these pericellular nets we have traced back to the great superficial petrosal nerve, in which they constitute small myelinated fibers of typical preganglionic character. Postganglionic fibers leave the sphenopalatine ganglion with the various nerves which radiate from it.

Large sensory fibers from the great superficial petrosal nerve pass in considerable numbers through the sphenopalatine ganglion directly into the palatine nerves and probably into other branches, with which they are presumably distributed to the palate and elsewhere. (Fig. 6.) Bundles of unmyelinated fibers, and also of medium sized myelinated fibers, possibly sensory, also enter from the deep petrosal part of the vidian nerve. (Figs. 7 and 8.)

We find the sphenopalatine branches of the maxillary nerve passing through the sphenopalatine ganglion without interruption into the palatine nerves, confirming the observations of Rhinehart in the mouse.<sup>36</sup>

Among the numerous components of the facialis in lower vertebrates, which have been accurately mapped by morphologists, we recall those somatic afferent fibers which reach the skin region involved in development of the external ear.<sup>24 25 30 31 37</sup> In the mouse, Rhinehart<sup>36</sup> has demonstrated this component supplying the skin of the back of the ear; he has also traced a few fibers through the ramus auricularis vagi, supplying part of the external auditory meatus and the tympanic membrane. Otherwise, this region is abundantly supplied by fifth nerve connections as mapped out by Gordon Wilson. The clinical studies of Ramsay Hunt<sup>16 17 18</sup> in herpes oticus are strong evidence of the existence of the ramus cutaneus facialis of Rhinehart in man, although it has not been demonstrated hitherto in the adult. Originally contested by Mills<sup>38</sup> and Wilson,<sup>39 40</sup> and lately by Key-Aborg, the findings of Hunt have received clinical confirmation from numerous observers<sup>42 43 44 45 46</sup> during fifteen years.



We have found a small branch corresponding very closely to Rhinehart's *ramus cutaneus facialis* in a human fetus of 54 mm. crown-rump length; this branch joins with the auricular branch of the vagus. (Fig. 9.) Such a joining between the *facialis* and the auricular branch of the vagus is described and figured by many anatomists. Presumably these facial fibers are distributed as in the mouse, namely, through the *ramus auricularis vagi* (Fig. 10), to supply part of the external auditory meatus, the tympanic membrane, and the skin of the cranial side of the concha. (Fig. 11.) In our series this branch is given off close to the *chorda tympani* but a little distal to the latter. Still more distally we have found another branch of the *facialis* which may correspond to the cutaneous filaments of the facial nerve to the auricle described by Valentin<sup>47</sup> in the adult human. In the embryo this branch is directed toward the auricle but could not with certainty be traced to the surface.

The palatine branch of the facial, considered as visceral sensory in lower vertebrates,<sup>41</sup> has its morphologic equivalent in the great superficial petrosal nerve in man. After the development of the sphenopalatine ganglion, in forms above the fishes, preganglionic fibers to this ganglion also become a component of this nerve. It will be recalled that both van Gehuchten<sup>14</sup> and Yagita<sup>15</sup> caused degeneration of geniculate ganglion cells, the former by section of the emerging seventh at the stylo-mastoid foramen, the latter by section of the great superficial petrosal.

It is now agreed that the geniculate ganglion belongs to the craniospinal series of sensory ganglia, and that its cells are of the unipolar sensory type.<sup>33, 48</sup> We have found in the great superficial petrosal nerve numerous large myelinated fibers of the sensory type, presumably the same as those cut by Yagita<sup>15</sup> and mentioned by Müller and Dahl.<sup>34</sup> We find also numerous 2—4  $\mu$  small myelinated fibers corresponding to preganglionic autonomic fibers elsewhere in the body.

In a two weeks' kitten (Fig. 5), we have found unmyelinated fibers, both small and large. The small fibers are post-ganglionic, from scattered groups of ganglion cells along the

nerve. The larger fibers may, in so young an animal, represent sensory fibers which have not yet acquired a myelin sheath. Our study of human material leads us to accept Rhinehart's conclusions on the mouse, that "both the chorda tympani and the great superficial petrosal contain afferent fibers whose cell bodies are located in the geniculate ganglion, and efferent fibers which pass through the ganglion without connection with its cells."<sup>36</sup>

General visceral afferent fibers, distributed through most of the branches of the primitive facialis,<sup>40</sup> would, therefore, be found also in those rami passing through the sphenopalatine ganglion. It will be recalled that we have demonstrated large myelinated sensory fibers from the palatine nerves, passing through the sphenopalatine without interruption to the cells of Yagita in the geniculate ganglion.

Here also in the geniculate are found the ganglion cells of Rhinehart's cutaneous auricular branch of the facialis. (Fig. 12.) We have, therefore, demonstrated the usual conditions for reference of pain from a visceral region to a body surface, in that ganglion cells of afferent fibers of both are located in proximity in the same sensory ganglion, while centripetal processes from both sets of cells pass together into the bulb.

Vasomotor changes, infiltration, infection or pressure in the sphenopalatine sensory distribution stimulates the palatine seventh and other visceral sensory fibers. Such impulses, passing through the great superficial petrosal nerve to the geniculate ganglion, come into relation with the somatic sensory cells and fibers of the ramus cutaneus facialis, transferring the pain impulse to the auricular and mastoid region.

This study is anatomic, and we have in no sense undertaken to discuss theories of referred pain. Anatomic solution of this problem seems still far out of reach. As we have recently stated,<sup>22</sup> "If one accepts the modified conception, based on the histologic studies of Dogiel, now held by many, that reference of pain from visceral organs to body surfaces is due to transference of the impulse from the afferent visceral fibers to somatic ganglion cells in the dorsal root ganglion, or its equiva-

lent, the relation of the ramus cutaneus facialis to the geniculate ganglion furnishes the necessary anatomic conditions. If, on the other hand, reference of pain should prove to be due to anatomic or physiologic relations of visceral and somatic afferent fibers within the central nervous system, the conditions for this would seem to obtain in the connections of the nerves under consideration within the medulla oblongata."

Much earnest work is being done on the craniocervical sympathetic system,<sup>9 11 50</sup> but it is to be hoped that less of theory and speculation and more of experimental and microscopic evidence will be produced. Remembering the fantastic and abandoned ideas of Fliess,<sup>51</sup> Bonnier<sup>52</sup> and others, we urge attention to Halphen's recent caution:<sup>10</sup> "Aujourd'hui, la mode est au sympathique, et nous croyons posséder la vérité parce que nous rattachions au sympathique ce que nous rattachions hier à la anaphylaxie, avant-hier à des reflexes bulbaires."

With Bürger,<sup>21</sup> we question the soundness of speculation or of empirical therapy in this field of referred pain unless and until definite anatomic pathways can be established; and we shall hope to present the results of further neural study in subsequent communications.

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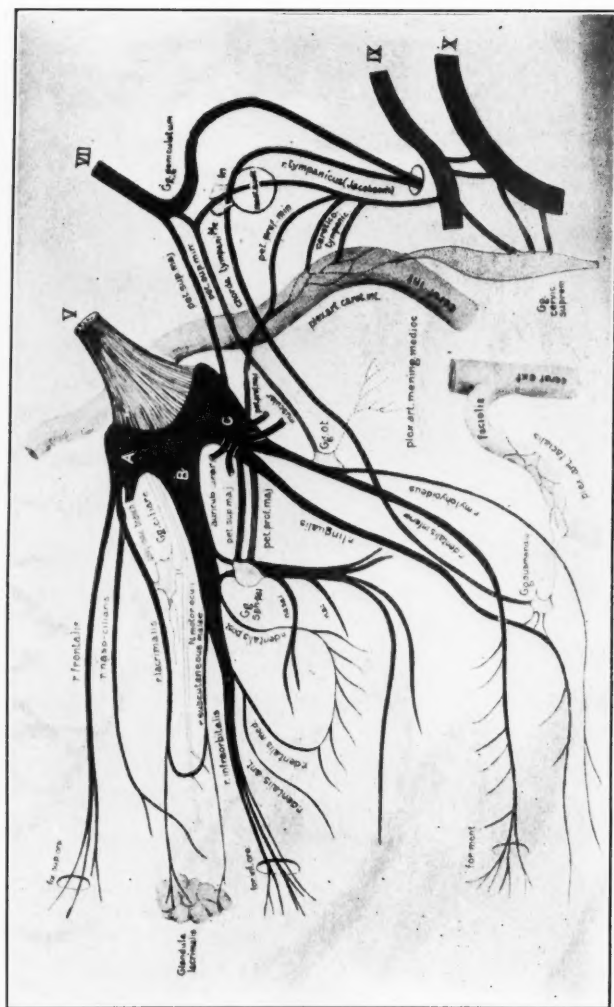


Fig. 1. The interrelation of the trigeminals, facials, glossopharyngeals and vagus, together with the sympathetic ganglia in man. (From H. H. Wilder: *History of the Human Body*; after Gray, Arnold and Gegenbaur.)



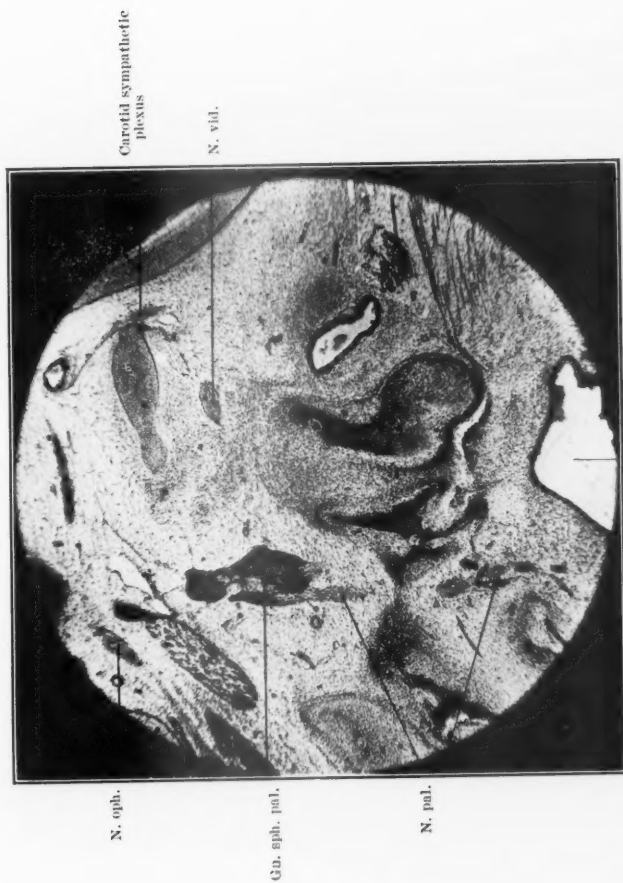


Fig. 2. Photomicrograph showing section of sphenopalatine ganglion, palatine nerve and Vidian nerve. Human embryo 54 mm. C. R., Hem. and Eosin, Parasagittal section; *gn.sph.pal.*, sphenopalatine ganglion; *n.opht.*, palatine nerve; *n.vid.*, nerve of Vidian canal; *n.opht.*, ophthalmic nerve. (Larsell.)

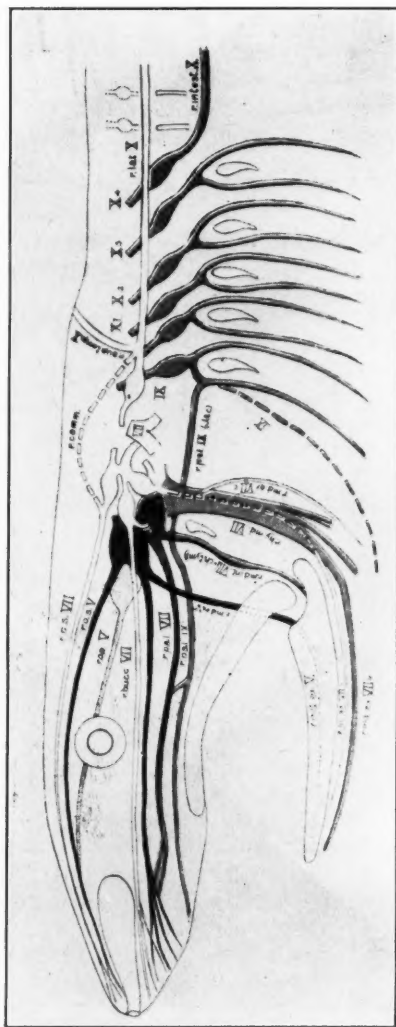


Fig. 3. Diagram of cranial nerves in *Anamnia*. (From Wilder, after Wiedersheim.) Roman numerals indicate the cranial nerves. Branches of these are designated as follows: *r.o.s.*, ramus ophthalmicus superficialis; *r.o.p.*, ramus ophthalmicus profundus; *r.bucc.*, ramus buccalis; *r.pal.*, ramus palatinus; *r.md.ext.*, ramus mandibularis externus; *r.md.int.*, ramus mandibularis internus; *r.hy.md.*, ramus hyomandibularis; *r.lat.*, ramus lateralis; *r.intes.*, ramus intestinalis.

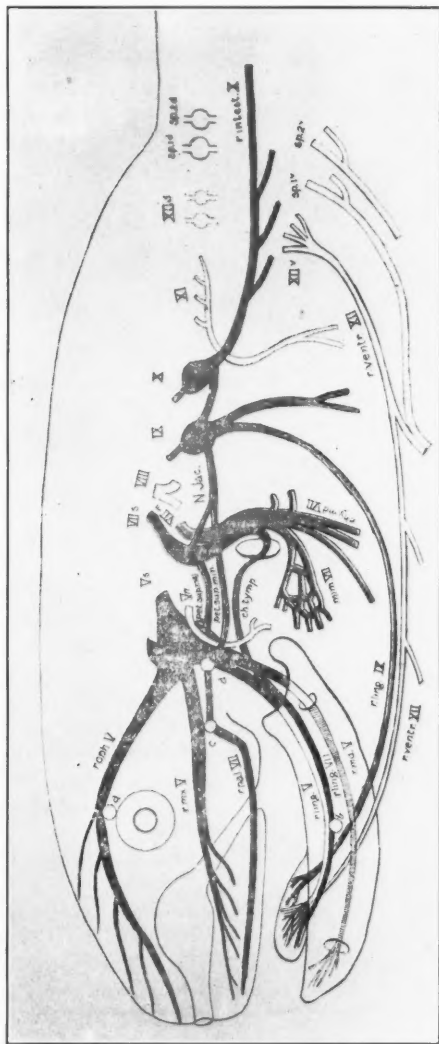


Fig. 4. Diagram of cranial nerves in *Amniota*. (From Wilder, after Widersheim.) Roman numerals indicate the cranial nerves; *sp-1* and *sp-2* indicate first and second spinal nerves. Branches as follows: *r.op.h.*, ramus ophthalmicus; *r.m.x.*, ramus maxillaris; *r.pal.*, ramus palatinus; *ch.tymp.*, chorda tympani (ramus mandibularis internus); *r.v.l.g.*, ramus lingualis; *r.m.d.*, ramus mandibularis; *min.*, mimetic nerve; *r.h.y.m.d.*, ramus hyomandibularis; *r.centr.*, ramus ventralis; *bol.sup.maj.*, petrosus superficialis major; *bol.sup.min.*, petrosus superficialis minor; *n.jac.*, Jacobson's nerve; exponents *d* and *v* denote dorsal and ventral roots; *s* and *m* denote sensory and motor branches; *a.*, ciliary ganglion; *b.*, submaxillary ganglion; *c.*, phenopalatine ganglion; *d.*, otic ganglion. Systems as in Fig. 2.

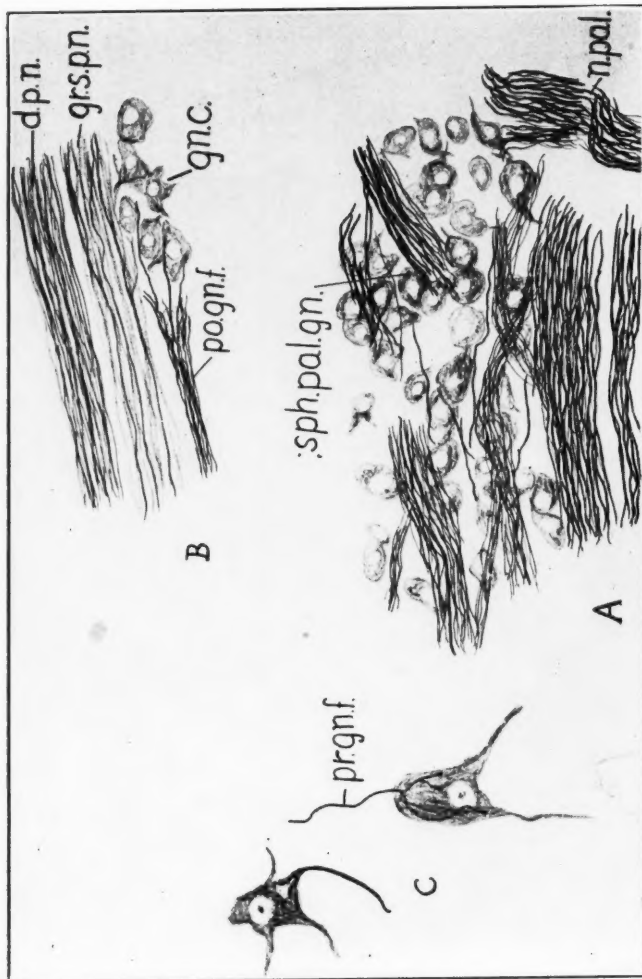
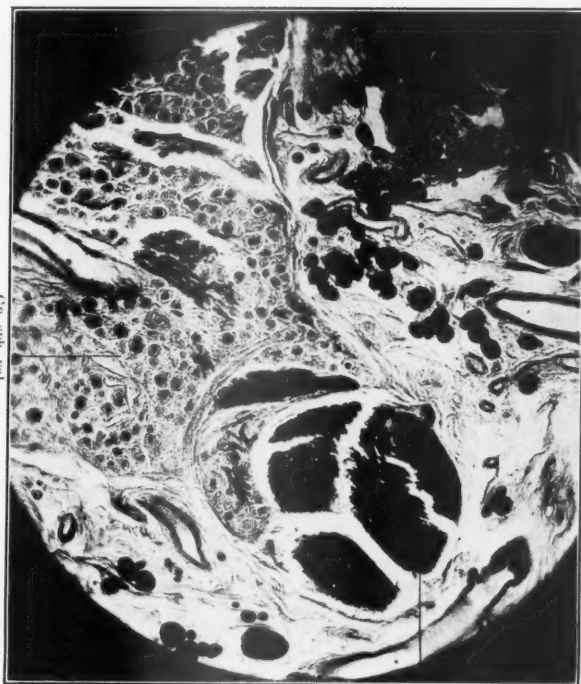


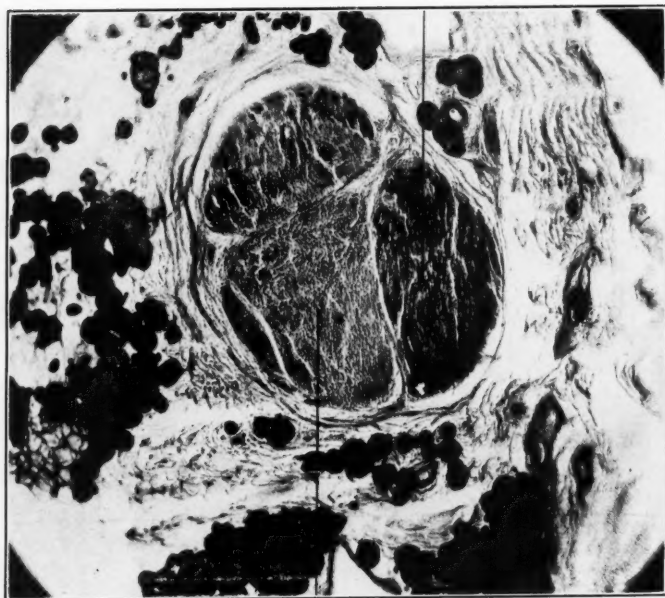
Fig. 5. A, Portion of the sphenopalatine ganglion. B, Portion of Villan nerve, with small cluster of ganglion cells. C, Sphenopalatine ganglion cells; *gr.s.pn.*, great superficial petrosal nerve; *n.pal.*, palatine nerve; *pog.nf.*, postganglionic fibres having their origin from the ganglion cells; *pr.gn.f.*, preganglionic fibre terminating in and not about ganglion cell; *sph.pal.gn.*, sphenopalatine ganglion. Kitten of two weeks. Pyridinesilver technique. Camera lucida drawings. A and B  $\times 470$ ; C,  $\times 960$ . (Larsell, in Trans. Am. Otol. Soc., 1928.)

Gn. sph. pal.



N. pal.

Fig. 6. Section of sphenopalatine ganglion and palatine nerve as latter is emerging from the ganglion. Adult human. Osmic acid stain *n.pal.*, palatine nerve. (Larsell.)



N. petr. prof. maj.

N. petr. sup. maj.

Fig. 7. Section of Vidian nerve. Adult human. Osmic acid stain. *n.petr.sup.maj.*, great superficial petrosal nerve; *n.petr.prof.maj.*, great deep petrosal nerve. (Larsell.)

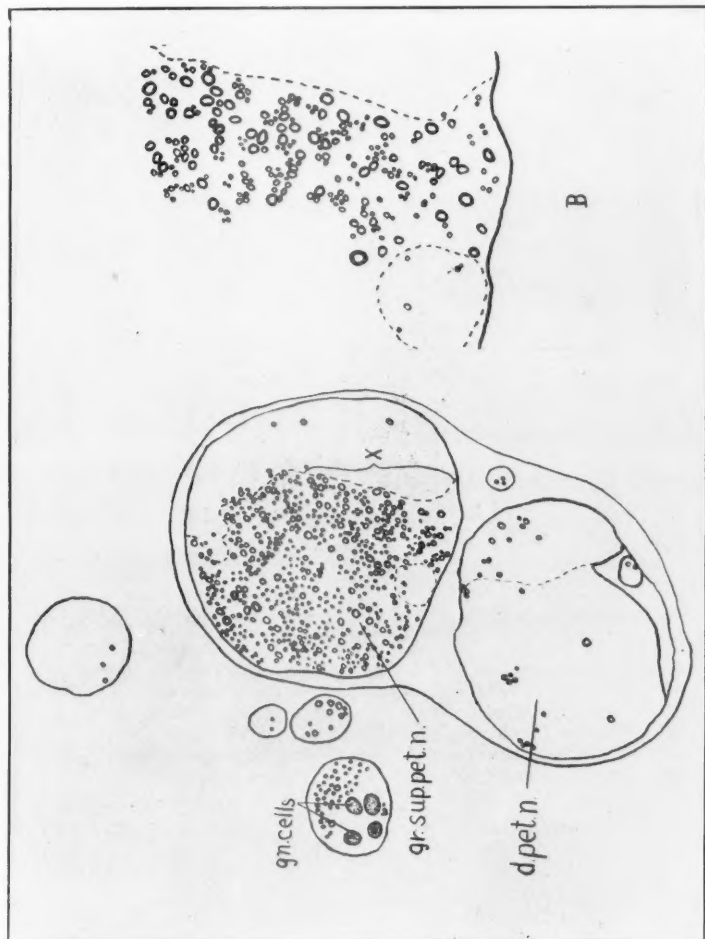


Fig. 8. A. Section of the nerve of the Vidian canal showing myelinated nerve fibres. The fibres of large diameter are of sensory type; those of small diameter are preganglionic fibres to the sphenopalatine ganglion. B. Longitudinal section of the nerve showing myelinated nerve fibres. The fibres of large diameter are of sensory type; those of small diameter are preganglionic fibres to the sphenopalatine ganglion. In osseous acid within 24 hours after death. *gn cells*, ganglion cells; *gr sup pet n.*, great superficial petrosal nerve; *d pet n.*, deep petrosal nerve. Fig. 8. A. drawn with aid of Edinger-Letz projection apparatus; Fig. 8. B. drawn with camera lucida. (Larsell in Trans. Am. Otol. Soc., 1928.)



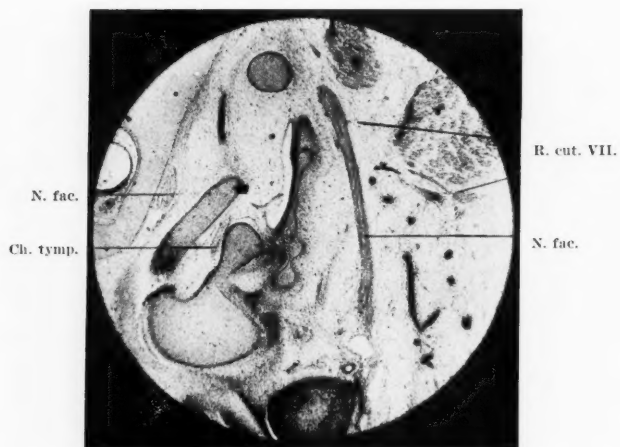


Fig. 9. Photomicrograph showing portions of facial nerve, chorda tympani, auricular branches of vagus, and communication of facial nerve with vagus (cutaneous branch of facial). Human embryo 54mm. C. R., Hem. and Eosin. Parasagittal section. *n.fac.*, facial nerve; *ch.tymp.*, chorda tympani; *r.aur.X.*, auricular branch of vagus; *r.cut.VII.*, cutaneous branch of facialis. (Larsell.)

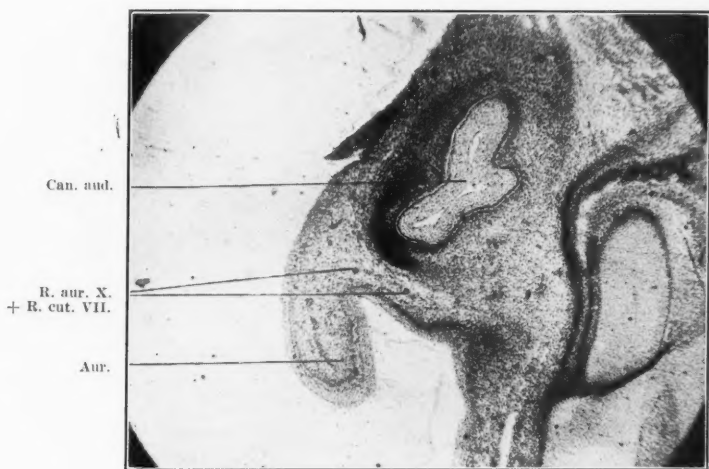


Fig. 10. Photomicrograph of section through auricle and external auditory meatus, showing auricular branches of vagus and facial nerves. Human embryo 54 mm. C. R., Hem. and Eosin. Parasagittal section. *aur.*, auricle; *m.aud.*, auditory meatus; *n.aur.X.+ cut.VII.*, auricular branches of vagus and facialis. (Larsell.)

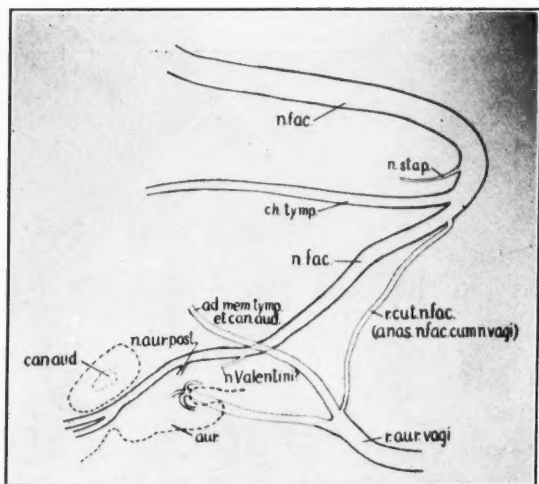


Fig. 11. Reconstruction on one plane from serial sections of facial nerve and its principal proximal branches, showing cutaneous branch of facialis anastomosing with auricular branch of vagus. Human embryo 54 mm. C. R.; *n. fac.*, facialis nerve; *n. stap.*, stapedius nerve; *ch. tym.*, chorda tympani; *r. cut. fac.*, cutaneous branch of facialis; *r. aur. vagi.*, auricular branch of vagus; *aur.*, auricle; *can. aud.*, external auditory canal; *n. aur. post.*, posterior auricular nerve; *n. Valentini*, cutaneous nerve of Valentin. (Larsell.)

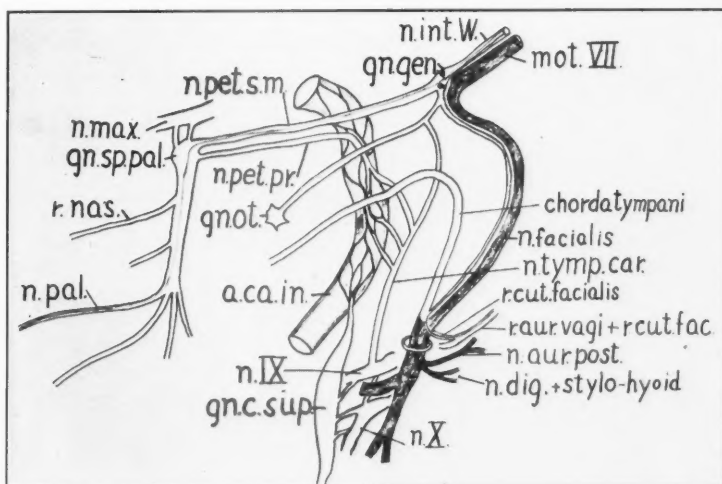


Fig. 12. Diagram of sphenopalatine ganglion and its connections, showing probable pathway of pain in otalgia from involvement of sphenopalatine region. Motor portion of facialis shaded. *a.c.a.in.*, internal carotid artery; *gnc.s.snp.*, superior cervical ganglion; *gu.ot.*, otic ganglion; *mot.VII.*, motor root of facialis nerve; *n.aur.post.*, posterior auricular nerve; *n.dig.stylohyoid.*, nerve to posterior belly of digastric and to stylohyoid muscles; *n.int.W.*, intermediate nerve of Wrisberg; *n.max.*, maxillary nerve; *n.pal.*, palatine nerve; *n.pet.pr.*, deep petrosal nerve; *n.tymp.car.*, caroticotympanic nerve; *n.nas.*, nasal nerve; *n.X.*, vagus nerve; *n.IX.*, glossopharyngeal nerve. (Larsell in Trans. Am. Otol. Soc., 1928.)

## LX.

### PRIMARY CARCINOMA OF THE BRONCHI.\*

BY LEROY A. SCHALL, M. D.,

BOSTON.

"Primary tumors (of the lungs) are rare."—Osler.<sup>1</sup>

"Tumors of the lungs are rarely primary."—Coplin.<sup>2</sup>

"Primary carcinoma of the lungs and pleura is considered relatively rare."—Tice's Medicine.<sup>3</sup>

"Primary carcinoma of the lungs is an uncommon disease."—Oxford Medicine.<sup>4</sup>

"Lung tumors are infrequently diagnosed antemortem."—Nelson's Medicine.<sup>5</sup>

Primary carcinoma of the lungs is by no means as rare as the textbooks might lead one to suppose. Previous to the last decade the diagnosis has usually been that of the autopsy table. Even the autopsy statistics show a wide variation as to the frequency of primary new growths in the lungs. Lord<sup>6</sup> found but eight instances of primary lung cancer in 4,704 autopsies at the Massachusetts General Hospital, but collected eight others. Weller<sup>7</sup> reports forty cases in 11,903 necropsies. Reinhard,<sup>8</sup> five in 8,716 autopsies, while Carman<sup>9</sup> states it occurs in 1 per cent of all cancers and over 2 per cent of all deaths from pulmonary disease. Eloesser<sup>10</sup> found it occurring in 3.9 per cent in 206 necropsies, and 20 per cent of forty cancer autopsies. Briese,<sup>11</sup> among 1,287 cases with cancer at autopsy, reports sixty instances of primary bronchopulmonary cancer, this site occupying sixth place in point of frequency. McCrae, Funk and Jackson<sup>12</sup> recently confirm Barron's<sup>13</sup> impression

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The X-rays illustrating this article were taken by Dr. A. S. Macmillan, roentgenologist of the Massachusetts Eye and Ear Infirmary.

The biopsy and autopsy reports were made by Dr. Tracy B. Mallory, pathologist of the Massachusetts General Hospital.

that the disease is increasing in frequency. Whether it is an actual increase or a relative increase due to increased interest and more accurate diagnosis is problematical. The theory that the pulmonary trauma suffered during the influenza epidemic of 1918-1919 may be responsible for an actual increase appears reasonable.

Most authors agree that histogenetically there are three sites of origin: (1) The bronchial mucous membrane; (2) the bronchial mucous glands, and (3) the alveolar epithelium. Paessler,<sup>14</sup> however, claims that no definite case has yet been shown to have originated from the alveoli. Both Adler<sup>15</sup> and McCrae<sup>12</sup> emphasize the fact that a primary carcinoma of the lungs is a bronchial carcinoma.

Histologically, tumors arising from the bronchial epithelium are either squamous or cylindrical cell tumors, and those from mucous glands are mainly adenomatous in structure, although atypical varieties may occur.

Males are more frequently affected. The age group is past 40 years. Approximately 91 per cent of McCrae's cases were past 35 years of age. Adler collected six cases between the ages of 10 and 20 years. One of my cases was 19 years of age.

The symptoms of cough, dyspnea, pain, hemoptysis, fever and cachexia all vary in degree and combination, depending upon the size and location of the growth. Cough is the most constant symptom. It usually grows progressively worse, although there may be periods of comparative freedom. Dyspnea often occurs early and is increased by exertion. The pain is described as being "deep in the chest." Hemoptysis may occur early, it may be slight in amount, merely tinging the sputum, or it may be severe. In the early stages fever is absent. Later, if bronchiectatic or abscess cavities develop, the temperature may be high, due to the secondary infection. Cachexia occurs late and usually there is a marked loss of weight.

In the differential diagnosis, tuberculosis, pulmonary abscess, gangrene, cyst, foreign body and aneurysm must be considered. Perhaps the most frequent error is that of tuberculosis. With the variable lung signs and with negative sputum examinations the error should be avoided. It is important to remember, however, that there may be a double process. Rakitansky<sup>16</sup>

at first insisted that tuberculosis and cancer were incompatible diseases, but later admitted that cancer might follow tuberculosis, but tuberculosis does not follow cancer.

From the viewpoint of the roentgenologist, Childs<sup>17</sup> states that tuberculosis masses usually are found in the posterior mediastinum, while cancerous nodules are more frequently in the anterior mediastinum. Carman<sup>9</sup> finds that cases erroneously diagnosed fall into two groups: (1) Those mistaken for mediastinal tumor, bronchopneumonia, gangrene, tuberculosis, cyst or empyema, and (2) those in which the pulmonary tumor is latent, and symptoms of extrathoracic metastases predominate. Although McMahon and Carman<sup>18</sup> believe the roentgen ray shows diagnostic characteristics, Holmes and Hyde<sup>19</sup> conclude that rarely the roentgenogram findings present features that are pathognomonic. It is quite evident that a bronchial new growth that produces chest changes demonstrable to the X-ray, unless these changes are secondary to bronchial occlusion, is in an advanced stage of the disease.

The diagnosis rests mainly on the bronchoscope. The bronchoscopic picture is that of bronchial occlusion, either by a tumor mass, outcropping or stenosis due to infiltration of the bronchial wall with smooth bronchial mucosa. This latter type of case may require repeated bronchoscopies before the tumor invasion is evident. Although the picture is fairly characteristic, biopsy makes the diagnosis positive. It is our belief that every obscure chest condition should have the benefit of a diagnostic bronchoscopy. At the thoracic clinic of the Massachusetts General Hospital every obscure chest condition, after thorough study in the wards, is seen by the internist, the thoracic surgeon and the bronchoscopist, as a group, and we feel that only by such close cooperation can an early diagnosis be made.

#### CASE REPORTS.

Case 1.—L. S., a housewife, aged 19, complained of cough and pain in the left side of the chest for five months. Twice she expectorated a teaspoonful of blood. There was slight improvement, but in a few weeks the cough and pain returned. There is no sweating but slight loss of weight. The



cough is mostly in the morning with but little expectoration. There has been progressive weakness.

X-ray report (January 11, 1927): There is no normal lung visible on the left. The lower two-thirds of the chest is dull. In the upper and outer portion there is a bright area which suggests gas either in the pleural space or in a cavity. The diaphragm is not visible on this side. The heart and mediastinal contents are displaced towards the left. The right lung appears normal. The appearance could be due to distension or collapse of the left lung with air in the pleural space.

Bronchoscopy (December 30, 1926): Under local anesthesia the 7 mm. Jackson bronchoscope readily passed into the left primary bronchus, where a definite, rounded, nonulcerative tumor mass completely filled the bronchus and moved readily upon respiration. A small specimen was removed for pathologic examination. The bronchoscopic diagnosis of carcinoma of the left primary bronchus was confirmed by the biopsy.

Autopsy (January 16, 1927)—Bronchi: The left primary bronchus shows a cylindrical tumor mass about 2 cm. long and 1.2 cm. in diameter, completely filling the lumen. It is adherent only at the base. It is rather white and soft, without evidence of necrosis.

Bronchial glands: There is one large fluctuant gland just below the bifurcation of the trachea. It contains reddish, semipurulent material and there is no evidence of metastasis within it.

Lungs: The right lung shows a compensatory emphysema and considerable hypostasis of the dependent parts. The left lung consists only of a sac of pleura in which the remains of dilated bronchi and fibrous interlobular septa are left suspended in masses of purulent material. No lung parenchyma whatever is left.

Microscopic examination—Tumor of the bronchus: This is composed of well differentiated epithelial cells. They are cuboidal, the nuclei round or oval with prominent chromatin granules and limiting membrane. There are no squamous cells or tendency to form epithelial pearls. The cells are arranged in cords and masses with a tendency to grouping in circular clusters about the stroma, suggestive of cylindromas. A single section from the base of the tumor where it is infiltrating the

lung shows a much more atypical structure with an occasional mitosis. (Fig. 1.)

Diagnosis: Carcinoma.

Case 2.—M. Z., a laborer, aged 41 years, had a cough which began one year previously and which, for the past seven months, has been productive. There has been hemoptysis varying from merely blood tinged sputum to two ounces of blood. For four months he has had chills and fever with increasing dyspnea. For two weeks there has been a sharp shooting pain "deep" in the chest on both sides of the sternum. The patient has lost 29 pounds in weight in the past four months.

X-ray report: Complete obliteration of the right lung field, the process excluding air from the right lung, consistent with neoplasm. (Fig. 2.)

Thoracic clinic: "Probably malignant lesion. We suggest bronchoscopy, as there is a possibility of other than malignant disease."

Bronchoscopy (July 28, 1927): Under local anesthesia the larynx was exposed and found to be twisted with a slight deviation to the left. The 9 mm. Jackson bronchoscope was passed through the cords, and in the region of the carina there was a large mass of ragged, ulcerating tissue which completely blocked the right main stem bronchus, with outcropping into the left bronchus. A small specimen was removed, followed by considerable bleeding, which was controlled by the use of sponges. Bismuth subcarbonate powder was insufflated against the growth for pneumography, the bronchoscope removed, and three X-ray plates taken. In rising to get off the table the patient suddenly became cyanotic and dyspneic. A second bronchoscopy was immediately done, inserting the bronchoscope past the tumor into the left main stem bronchus, which was found partly filled with blood. The blood was aspirated, and by means of artificial respiration the breathing was re-established. After waiting fifteen minutes with normal respiration and no bleeding it was thought safe to remove the bronchoscope. Upon removing the bronchoscope again the respiration ceased; the bronchoscope was again inserted down the left main stem bronchus. There was no bleeding. Artificial respiration failed to reestablish breathing and the heart action stopped.

Adrenalin, 1/1,000 5 cc. was injected directly into the heart muscle without effect.

The biopsy report confirmed the bronchoscopic diagnosis of carcinoma.

Case 3.—Mrs. P. T., aged 33 years, was seen with and is reported through the courtesy of Dr. D. Campbell Smyth. Dr. John Sproull, of Haverhill, Mass., in referring the patient, gave us the following history:

"I saw Mrs. T. for the first time October 26, 1926, when she was having fresh air and rest cure for supposed pulmonary tuberculosis. She gave a history of having been ill for ten months, her illness having begun with a dry, unproductive cough, for which she consulted a physician, who advised her to have her tonsils out. The operation of tonsillectomy was performed without a preliminary physical examination of the chest. After the operation her cough became very much worse and she raised a little mucoid blood tinged sputum. She consulted another physician, who examined her, and said she had pulmonary tuberculosis. From that time on she suffered from severe coughing spells, attacks of left-sided pleuritic pain, some shortness of breath, weakness and occasional elevations of temperature. In August, 1926, the state tuberculosis examiner saw her in consultation and said she had a left-sided pleurisy with effusion, probably tubercular, and advised an X-ray, which was not done.

"Physical examination shows absence of breath sounds, flatness, absence of vocal and tactile fremitus all over the left chest, with the apex beat of the heart in the fourth interspace at the left axillary border. The temperature was 100° F. at night and normal in the morning.

"Twenty-five hundred cc. of clear, amber colored fluid was withdrawn from the left chest. Repeated sputum examinations have been negative for T. B. Blood Wassermann is negative.

"Diagnosis: Probable neoplasm of the left bronchus, and I am referring the patient for bronchoscopy."

X-ray report: A superradiant right lung field and free from any evidence of disease. The left lung field shows a marked homogeneous increase in density, through which no lung markings can be made out. The heart and mediastinum are markedly displaced toward the left. (Fig. 3.)

Bronchoscopy (March 15, 1927): Under local anesthesia the 7 mm. Jackson bronchoscope passed readily between the cords. The tracheal mucosa appeared normal. In the left primary bronchus, just beyond the carina, there is a large, ragged tumor mass, completely filling the lumen of the bronchus. A specimen was not taken.

Bronchoscopic diagnosis: Carcinoma of the left primary bronchus.

Autopsy (April 17, 1927).—Bronchi: The right bronchus is negative. The left bronchus is completely filled from a point of exit from the trachea by a cylindrical, white, firm tumor mass invading and thickening the walls. This point of origin appears to be at the first subdivision of the primary bronchus. From this point onward the entire bronchial tree is obliterated by a solid tumor extending throughout the left upper lobe of the lung, invading through the interlobar septum and via the subpleural lymphatics to the lower lobe, approximately half of which is involved in the process. The entire lung is shrunken and the remaining lung parenchyma collapsed. The left lung shows numerous small nodules in the pleura, averaging from 2 to 3 mm. in diameter, which are hard, white and umbilicated. A few similar nodules are present in the deeper portions of lung tissue. The tumor has extended through the root of the left lung to the paravertebral tissues and has completely encircled the aorta. From a point just beyond the arch, for a distance of 6 cm., the aorta has been markedly contracted, and at one point it is so narrow that it is impossible to pass the forefinger through it. (Fig. 4.)

Microscopic examination: An extremely scirrhus tumor in which the bands and cords of undifferentiated epithelial cells twist through a dense mass of fibrous tissue. For the most part the epithelial cells are undifferentiated. Mitotic figures are present but not numerous. In other areas the growth is more cellular and there is well marked differentiation into glands lined with cuboidal or cylindrical epithelium. Many of these cells show a bluish cytoplasm, sometimes vacuole formation, suggesting the possibility of mucous secretion. One section from the root of the lung shows invasion of the adventitia of the aorta with rapid spread of the tumor in the perivascular lymphatics of the vasa vasorum. (Fig. 5.)

Diagnosis: Primary carcinoma of the left bronchus.

Case 4.—G. F., a mechanic, aged 34 years, for the past six weeks has had pain and stiffness of the neck. A "wen," which he first noticed a few weeks previously on the right parietal region, was incised and "pus" obtained. Shortly after this he developed sharp shooting pains in both parietal regions; they radiate toward the vertex and are somewhat relieved by aspirin. About the same time the left shoulder was painful, and a "lump" appeared in the left axilla. Next, the right index finger became swollen and painful and the outer aspect of the left thigh is very painful. There have been no respiratory symptoms.

Physical examination shows a well developed and nourished man in discomfort from pain in the neck. There is a scalp dressing over the right parietal region. The neck is very painful on motion. The glands are pea to bean sized in the right cervical region. There is a hard subcutaneous nodule in the left axilla on the outer border of the pectoral muscle. There is slightly increased spinal dullness and increased subclavicular dullness on the left, the breath sounds are not obstructed and there are no rales.

X-ray report (September 16, 1927): Plates taken of the cervical and upper dorsal spine fail to show any definite variation from the normal. The bones of the thorax are also negative for metastases. The lower half of the left chest is less radiant than normal. The diaphragm on this side is a little high, and just above it, apparently obscured by the shadow of the heart, there is a round, dense area about 4 cm. in diameter, with fairly sharply defined margins. A similar smaller area is near the root of the lung. The appearance is consistent with malignant disease.

Autopsy (October 3, 1927).—Lungs: The pleural surfaces of all lobes of both lungs are covered with innumerable small, white, umbilicated, very firm nodules from pinhead to pea size. Similar small pearl like nodules are scattered throughout the lung parenchyma. The lower lobe of the left lung shows an egg shaped tumor 3.5 by 4.5 cm. This is directly continuous with a small tumor mass in the lining of a small bronchus of the third order, which seems unquestionably the primary focus of the tumor. In its outer portions the tumor has invaded the

base of the upper lobe, as well as spreading across the interlobar division, which has been partially obliterated by old fibrous adhesions. There is an enlarged lymph node immediately contiguous to the outer end of the tumor and lying just at the first division of the left primary bronchus. On cross section the tumor is for the most part of firm white, rather structureless tissue, but shows numerous opaque, yellowish, slightly caseous necrotic foci. At its primary focus in the bronchus it involves the mucous membrane in an annular ring for a width of 1.5 cm. The distal portion of the bronchus is definitely dilated, being almost equal in diameter to the left primary bronchus.

Diagnosis: Primary carcinoma of the left bronchus.

Case 5.—J. S., a sailor, aged 55, complained of an acute dry cough for the past six weeks. For three weeks there has been pain in the left lower chest. The cough has grown worse, accompanied by foul, yellowish green sputum, amounting to two to three cups a day. The sputum has been blood tinged for three days. The patient has felt weak and had lost 15 pounds in weight.

Physical examination: There are rales from the midscapula to the left base posteriorly and into the left axilla and anterior axillary line. Tactile fremitus and breath sounds are increased at the left base posteriorly. The breath sounds are diminished over the left side anteriorly.

X-ray report: Findings are those of a pneumonic process with fibrosis and incomplete expansion of the left lung.

Bronchoscopy (May 11, 1927): The 9 mm. Jackson bronchoscope, under local anesthesia, was passed through the larynx and in the left main bronchus, 2 cm. from the carina, a definite, pulsating, smooth obstruction of the bronchus was found projecting from the posterior medial surface. This obstruction was not complete, and by manipulating the bronchoscope the rest of the bronchus could be seen. There was no secretion. Fifteen cc. of lipiodol was instilled past the obstruction, the narrowed bronchus wiped dry and bismuth subcarbonate insufflated for X-ray study.

X-ray report (May 11, 1927): Stenosis of the left primary bronchus, 2 cm. from the bifurcation, evidently from external pressure. (Fig. 6.)

Interval history (September 24, 1927): The patient returned to work and thinks he gained 25 pounds in weight. For two or three weeks the sputum was slight in amount, with very little cough, but slight pain in the left axilla on exertion. Last week the cough increased and the sputum was blood tinged.

Second bronchoscopy (October 13, 1927): The 9 mm. Jackson bronchoscope was passed without resistance to the left main bronchus. Two cm. from the bifurcation a constriction was encountered which was dilated with forceps. Beyond the constriction there was a mass of tissue seen, resembling granulation tissue, and a piece was removed for microscopic examination.

Biopsy report: Two tiny fragments of tissue for microscopic examination show a cluster of hyperplastic, atypical, epithelial cells. Histologic appearance is that of carcinoma. (Fig. 7.)

Case 6.—P. F. M., aged 34 years, complained of having a cough and pain in the chest which began five months ago. One month later he had a sudden severe attack of coughing, with dyspnea, drenching sweats and a temperature of  $102^{\circ}$ . After two weeks of being confined to bed the temperature subsided, but the cough persisted, raising six ounces of thick, brownish, odorless sputum a day. The patient has grown steadily weaker and has lost 60 pounds in weight during the past four months. The left chest was tapped twice with negative results. Repeated sputum examinations have been negative for tuberculosis.

Physical examination shows a pale, cyanotic, wasted, sick-looking white male. The teeth and gums show pyorrhea. The posterior cervical, left supraclavicular and right epitrochlear glands are enlarged. The left lung is almost completely collapsed, with the heart drawn over to the left. The temperature is  $100.6^{\circ}$ , pulse 90, respiration 16, white blood count 12,000, red blood count 3,500,000.

X-ray report (October 25, 1927): The entire left chest is very dense. The heart and mediastinal contents are displaced to the left. Part of the lower right lobe extends beyond the midline. (Fig. 8.)

Bronchoscopy (October 29, 1927): Under local anesthesia the 9 mm. Jackson bronchoscope passed readily to the left

main stem bronchus. One cm. from the carina, the left main bronchus was completely occluded by a round, firm, nonulcerative growth, which mounded up into the lumen of the bronchus. A specimen was removed for biopsy.

The bronchoscopic diagnosis of carcinoma of the left bronchus was confirmed by the biopsy.

These six cases occurred within the past year. The diagnosis of carcinoma was made five times in some 200 bronchoscopies. This diagnosis was confirmed either by biopsy or biopsy and autopsy. One falls into Carman's second group with the extrathoracic metastases predominating and the primary lesion found only at necropsy.

#### CONCLUSIONS.

1. Primary carcinoma of the bronchus is not an extremely uncommon disease.
2. Patients with obscure chest conditions should have the benefit of close cooperation of the thoracic surgeon, the internist, the roentgenologist, and the bronchoscopist, acting as a group.
3. Bronchoscopy offers the best means of establishing an early diagnosis of malignancy of the bronchi.

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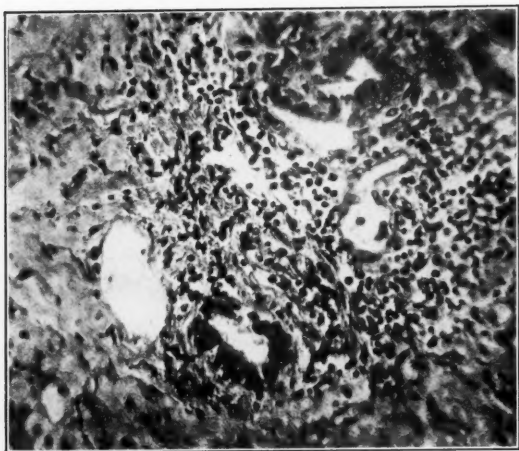


Fig. 1.

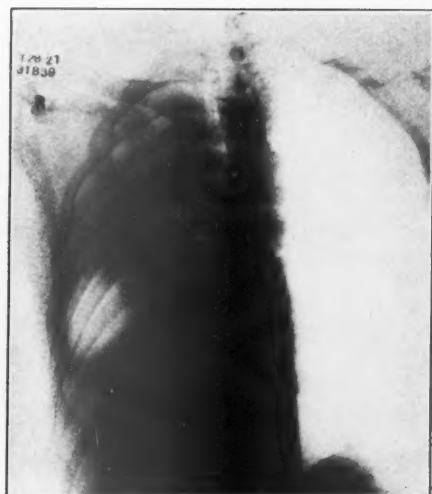


Fig. 2.



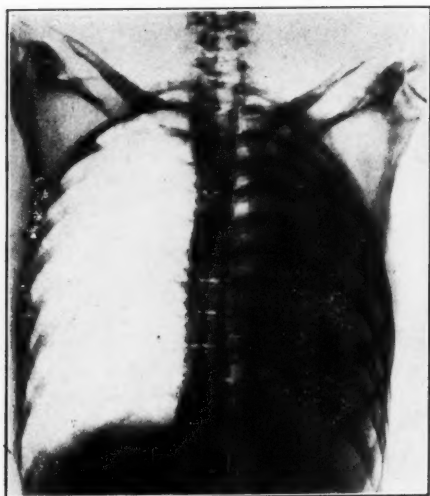


Fig. 3.



Fig. 4.





Fig. 5.



Fig. 6.



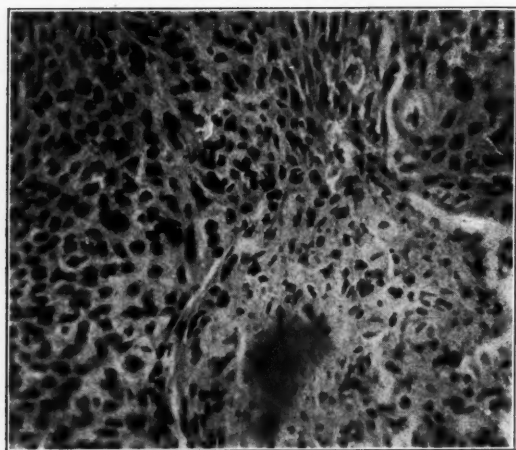


Fig. 7.

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Fig. 8.

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M.C.

LXI.

THE PRODUCTION OF OTITIS MEDIA AND LABY-  
RINTHITIS IN RABBITS.

By ERIC A. LINELL, M. D.,

MANCHESTER,

AND

H. H. BURNHAM, M. D.,\*

TORONTO.

Objects.—This series of experiments was undertaken with the main object of making a detailed study of the changes in the middle ear of the rabbit following experimental infection of the middle ear cavity. Small infections were given at short intervals because it was felt that information as to the pathologic changes in chronic otitis media would prove more valuable from the clinical point of view than a demonstration of the acutely inflamed middle ear cavity as it is seen on the operating table.

In a satisfactory percentage of animals infected we have produced a smouldering otitis media and in one case have been fortunate in obtaining a spread of infection to the labyrinth. It seems reasonable to hope that by this technic it may be possible to obtain an intracranial extension of the infection with the production of cerebral or cerebellar abscess.

Technic.—The rabbit is anesthetized with eucain and 2 to 3 cc. of a dilute broth culture of hemolytic streptococci is injected into the middle ear cavity through the tympanic membrane. A suitably sized ear speculum is placed in position in the external auditory canal and the organisms are injected through a long fine needle passed down the speculum. If fluid leaks back through the puncture hole in the drum, this is mopped up with absorbent cotton and the ear is left lightly plugged with the latter.

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The organism used was a hemolytic streptococcus, obtained from human scarlet fever cases, which was kindly supplied to us by Dr. Donald Fraser of the Connaught Laboratories of the University of Toronto.

The animal was, as a rule, off its food for one to two days after the operation, but thereafter it showed no ill effects whatever. It was interesting to note that the period required for recovery lengthened somewhat after each repetition of the injection, tending to show an increase of toxic absorption from an already damaged mucous membrane.

Otoscopic examination after operation was, on the whole, unsatisfactory. In some cases there was a little injection of the drum, which later tended to show some opacity, but these findings were not constant in our positive cases.

The injections were repeated at intervals of from two to four weeks, one rabbit having four infections of the right ear between the 23rd of December, 1924, and the 3rd of April, 1925.

Rabbit No. 2 had four infections of right ear.

Rabbit No. 4 had three infections of both ears.

Rabbit No. 5 had one infection of left ear and 2 infections of right ear.

A few weeks after the last infection the animal was chloroformed and the temporal bone was transferred to formalin fixation, care being taken to look for any signs of intracranial spread of inflammation. After decalcification the bone was cleared in oil of cedar, imbedded in paraffin and cut in serial horizontal sections from above downwards. The sections were mounted, stained with hematoxylin and eosin and examined.

Normal Anatomy.—A considerable time was spent cutting and examining serial sections of normal rabbit temporal bones to familiarize ourselves with the details of normal structure before any pathologic investigation was undertaken.

The rabbit's middle ear is very suitable for the study we had in mind, as it is a large flask-shaped cavity, capable of holding a considerable amount of injection fluid. The arrangement of tympanic membrane, ossicles and internal ear structures is sufficiently in line with that of the human to justify deductions from one to the other.

In our histologic studies we paid particular attention to the regions of the oval and round windows, and particularly to the

structure of the setting of the footpiece of the stapes into the bony labyrinthine capsule. (Figs. 1 and 2.) With reference to the latter it was of interest to note the multilocular arrangement of the joint by which the stapes footplate slides in the oval window. The loculi are separated by fine partitions of fibrous tissue. A high power view of this joint is shown in Fig. 3, and the fibrous septa are well seen in Fig. 4, in which the joint has been accidentally torn at its inner (labyrinthine) side and the stapes slightly hinged outwards.

As regards the internal ear, it would appear from the literature that premortem intravascular injection of fixing fluid is necessary in order to get perfect fixation of delicate structures like the organ of Corti. However the "en masse" postmortem fixation we employed seemed adequate (Fig. 5.), and we were afraid of upsetting pathologic pictures by the premortem injection of fluids into the labyrinthine vessels.

After an examination of several rabbit ears by serial section, two normal human ears were cut, fixed and mounted by the same technic and were used for purposes of comparison.

It was then felt that we might proceed to the examination of ears infected by the method described above.

#### RESULTS—A. OTITIS MEDIA.

Rabbit No. 5.—Otitis media (left), following one injection; no result (right), following two injections.

Rabbit No. 4.—Otitis media (left), slight, following three injections; otitis media (right), following three injections.

Rabbit No. 2.—Otitis media and labyrinthitis (right), following four injections.

A positive inflammatory reaction is thus shown to have occurred in four out of the five infected ears. The discrepancies in results as shown in this table are within the limits of experimental error; for instance, we believe that on one occasion we overheated the broth culture before injection and possibly killed the organisms. Two points of importance are (1) that the percentage of positive results is high enough to justify the technic and (2) that repetition of infections shows proportionally increasing pathologic effects. Rabbit No. 2, after four injections, died of the labyrinthitis and meningitis produced.

**Osteitis.**—There was abundant evidence of inflammatory bone disease throughout the sections. All gradations of a chronic progressive osteitis could be found, from the earliest stages in the formation of a "flake" sequestrum under an inflamed but intact mucous membrane (Fig. 6) to a definite ulcer with carious bone at its base. (Fig. 7). Early caries of a bony septum in the middle ear cavity with lymphocytic infiltration of the mucous membrane covering each side of the septum was also observed.

As well as progressive destructive lesions of bone, attempts at repair can be found. Such an area shows dead bone, overlying which is a cellular mass consisting for the most part of young fibroblasts and plasma cells. Lymphocytes are absent, and the picture is one of attempted fibrous encapsulation of a sequestrum. Such a pathologic process is seen in Fig. 8, where a projecting spicule of dead bone, surrounded by a fibroblastic reaction, is in process of being thrown off into the middle ear cavity as a fibrous tissue covered sequestrum.

This technic, therefore, has a definite value in providing an easily controlled method of studying experimentally the intimate pathology of subacute and chronic inflammatory bone disease.

**Granulation Tissue.**—Masses of granulation tissue are found lying in the middle ear cavity. For the most part these are found attached to a carious area of a bone, but in one case the antrum was full of this tissue. In Fig. 9 is a mass of granulation tissue which, by serial sections, is seen to owe its origin to an area of necrosis of one crus of the stapes.

**Destruction of the Stapes.**—This bone seems to be frequently attacked by the inflammatory process, because destruction, partial or complete, of the crura is seen in three cases, Figs. 9, 10 and 11. In Fig. 10, one side of the joint cavity is being invaded by lymphocytes from the middle ear side, but in none of these had the infection penetrated the joint and reached the labyrinth.

#### B. OTITIS MEDIA COMPLICATED BY ACUTE LABYRINTHITIS.

The right ear of rabbit No. 2 was the only case where we were successful in obtaining a spread of infection beyond the confines of the middle ear cavity. As previously stated, this

animal had four infections of its right middle ear cavity between December 23, 1924, and April 3, 1925. It died eleven days after the last infection. It never recovered from the last operation, and it is reasonable to presume that the acute labyrinthitis produced was the cause of death. A terminal meningitis seems probable from the histologic evidence, but this was not sufficiently definite to make a dogmatic statement.

Autopsy revealed no marked increase in pressure of cerebrospinal fluid, there was no macroscopic evidence of meningitis, and the right petrous bone, as viewed from inside the cranium, showed nothing abnormal. The petrous bone was fixed, decalcified and cut in the routine manner previously described.

A low power view of this ear (Fig. 12) shows masses of granulation tissue in the middle ear cavity, particularly in the antrum. There is a very marked destructive process involving both the perilymphatic and the endolymphatic channels of the cochlea, all of which are more or less filled with inflammatory exudate. In marked contradistinction is the apparently slight affection of the utricle, and the semicircular canals are also comparatively free of infection. The cochlear nerve fibers are well stained and are seen streaming down through the modiolus to the internal acoustic meatus.

**The Path of Infection.**—The path of labyrinthine infection in this case has been through the oval and round windows. A study of Figs. 13 and 14 shows the stapes subluxated inwards with a marked inflammatory reaction on the outer (middle ear side) of the joint. The labyrinth behind the oval window is filled with inflammatory exudate. Fig. 14 shows particularly clearly the free passage for inflammatory products from the middle to the internal ear cavities.

Fig. 15 is taken through the round window, to show the continuity of inflammatory reaction on the middle ear and on the labyrinthine side of the destroyed membrane.

It is assumed then that the infection reached the perilymph by these two routes. Fig. 16 gives a high power view of the cochlea. The *scalæ tympani* and *vestibuli* contain inflammatory exudate. Reissner's membrane has been destroyed with consequent infection of the canal of the cochlea. The remains of the organ of Corti are visible and the nerve fibers streaming from it into the modiolus are picked out in black in the photograph.

With a higher power it is seen that this is due to the perineural lymphatic spaces being filled with lymphocytes, from which it is argued that the infection has been able to track by these spaces to the internal acoustic canal. This is confirmed by Fig. 17, which is taken through the internal acoustic canal (which is a shallow depression of the posterior face of the petrous bone in the rabbit). There is a flaky necrotic bone, surrounding which is a small round celled infiltration. The path of infection can thus be traced through the labyrinth to the internal acoustic meatus.

It is of great interest to note that the inflammatory process involved to an almost negligible degree the posterior (vestibular) compartments of the internal ear. Fig. 12 shows an apparently normal utricle and Fig. 17 includes a macula. The slight subendothelial small round celled infiltration of this structure is remarkable, particularly in view of its close proximity to the definite inflammatory reaction in the internal auditory canal.

Fig. 18 shows edema and small round celled infiltration under the macula of the saccule, but the cavity of the saccule is free from inflammatory products, and it seems possible that this amount of reaction may be due to a reflux of infection from the internal auditory meatus and up the perineural lymphatics to the nerve endings in the macula.

Alexander describes a connective tissue septum which traverses the perilymphatic space in a transverse plane and serves to cut off the perilymph surrounding the saccule and canal of the cochlea from that surrounding the membranous utricle and semicircular canals. This may act as a barrier to the backward spread of infection which has entered the perilymph of the scala tympani and scala vestibuli in this case. At any rate, this case seems to prove that an infection of the labyrinth may be confined to its anterior or auditory portion, and that infection of one portion of the labyrinth does not necessarily involve the destruction of the whole, even though such infection has reached the internal auditory canal and possibly involved the meninges. This important fact is borne out by observations of subtotal labyrinthitis in human patients.

We wish to acknowledge helpful advice and facilities generously granted to us by Dr. Oskar Klotz, of the Department of Pathology, University of Toronto.



Fig. 1. Low power microphotograph of middle and internal ear cavities of fetal dog, to show relations of oval and round windows to the labyrinthine cavities. Note the myxomatous tissue found in the cavities at this stage of development.





Fig. 2. Low power microphotograph, adult rabbit. Shows mode of fixation of footplate of stapes in oval window.



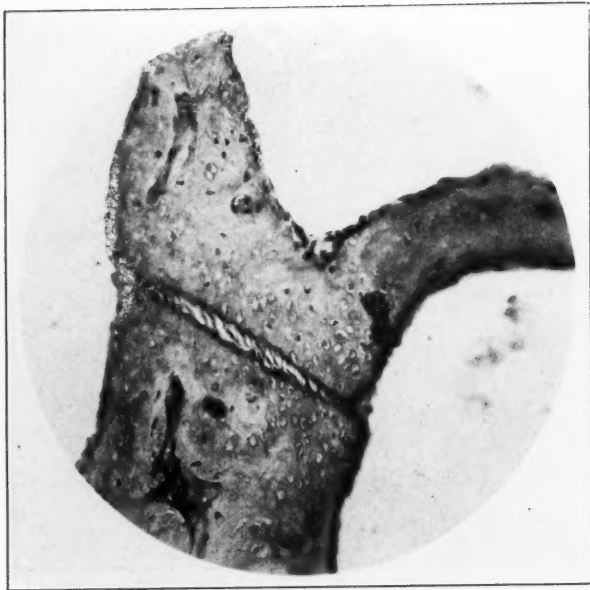


Fig. 3. High power view of stapedial joint, rabbit.



Fig. 4. Accidental rupture of joint. Shows the bundles of fine fibers which connect the opposing bony surfaces.





Fig. 5. Organ of Corti. Normal rabbit. Blood clot in scala tympani.

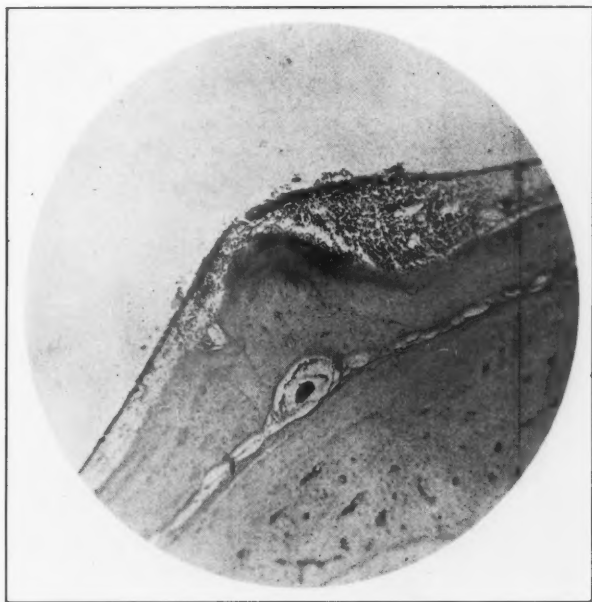


Fig. 6. Chronic otitis media. Rabbit. Section shows osteitis with lymphocytic infiltration of overlying mucous membrane.



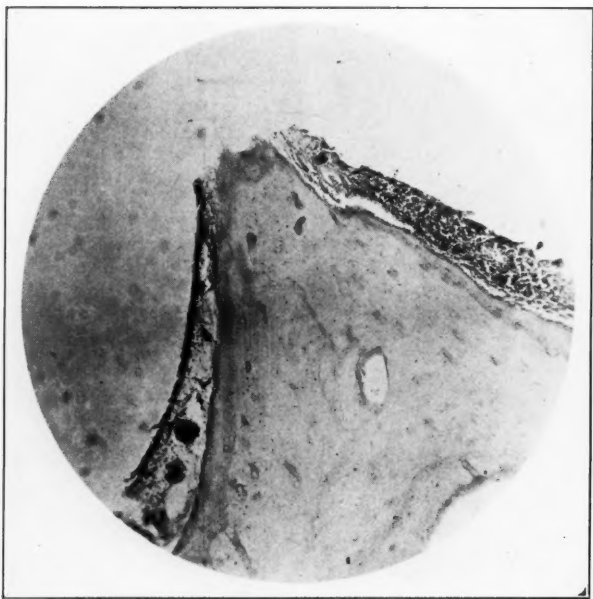


Fig. 7. Further stage to Fig. 6, with erosion of mucous membrane.  
Caries of exposed bone spicule.

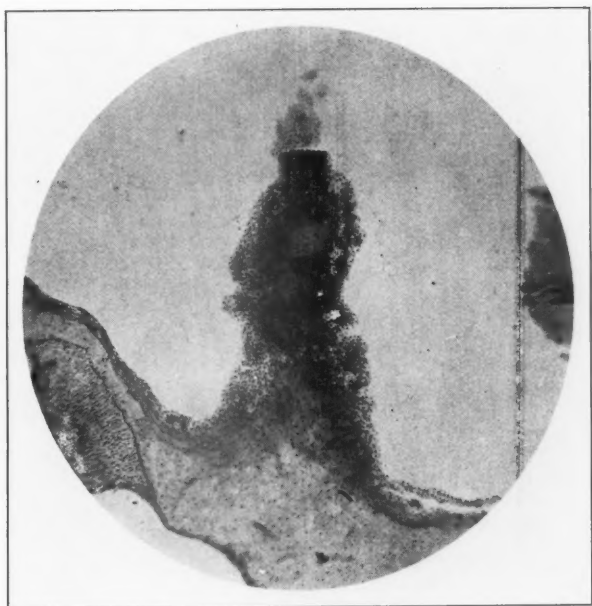


Fig. 8. Process of dead bone encapsuled by fibrous tissue and in process of being thrown off as a sequestrum. Rabbit. Middle ear infection.



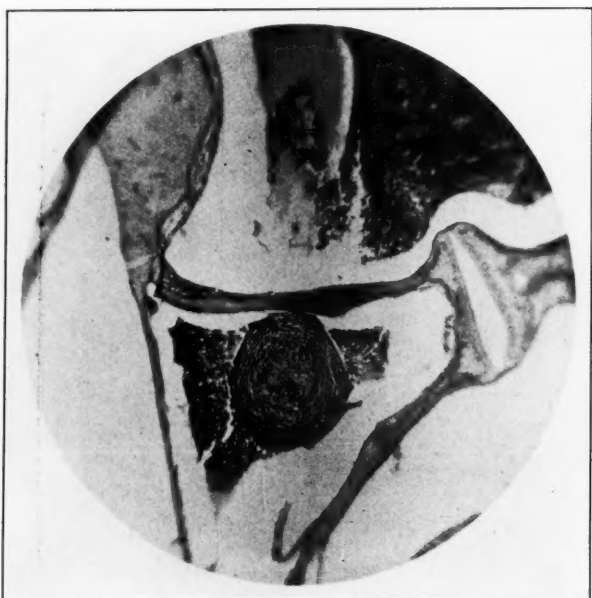


Fig. 9. Caries of crus of stapes. Granulation tissue lying between crura is attached to carious area of bone. Rabbit. Otitis media.





Fig.10. Destruction of crura of stapes by inflammatory process in middle ear. Note invasion of the stapedial joint by small round cells but no extension of inflammation to labyrinthine side of oval window.



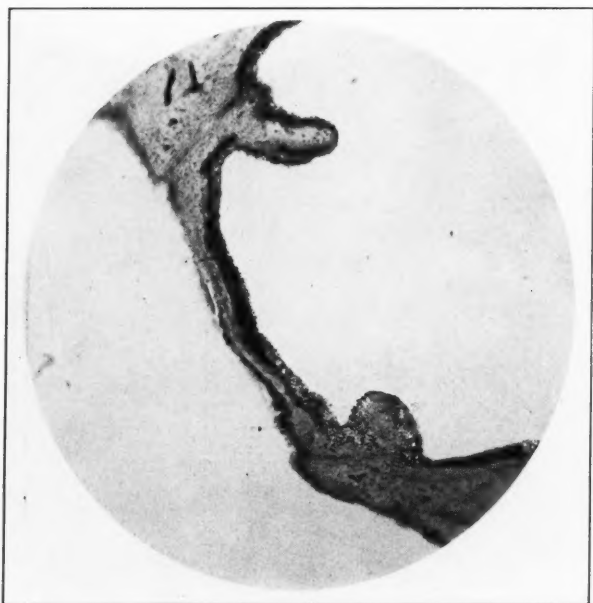


Fig. 11. Section from third case of this series showing caries of stapes. Almost complete destruction of the crura. No labyrinth invasion. Rabbit. Otitis media.

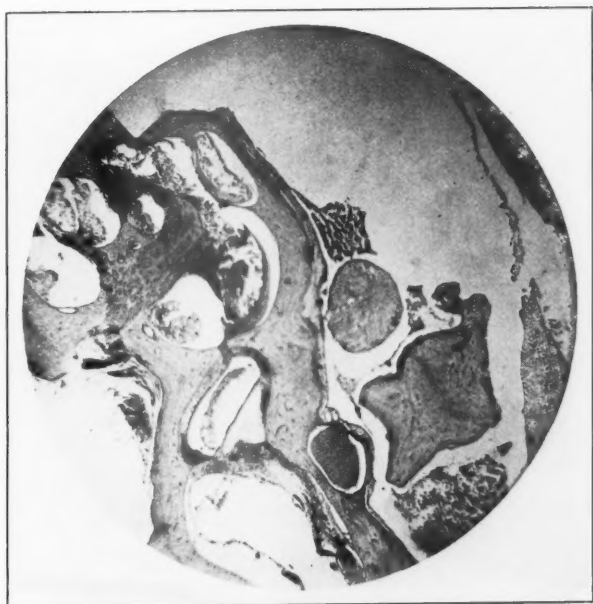


Fig. 12. Low power view of middle and internal ears of rabbit No. 4, showing granulation tissue in middle ear cavity and marked destruction of the cochlear part of the labyrinth. The cochlear nerve fibers can be followed to the internal acoustic meatus, picked out by the infiltration of their sheaths with lymphocytes. Note absence of inflammatory reaction in cavities of vestibular part of labyrinth.





Fig. 13. High power view of oval window and stapes in rabbit No. 4. Note subluxation of joint inwards, caries of crus, inflammatory reaction in middle ear and inflammatory exudate in labyrinth cavity.



Fig. 14. High power view taken through edge of oval window. Rabbit No. 4. Note the gap permitting inflammatory products to pass from the middle to the internal ear cavities.



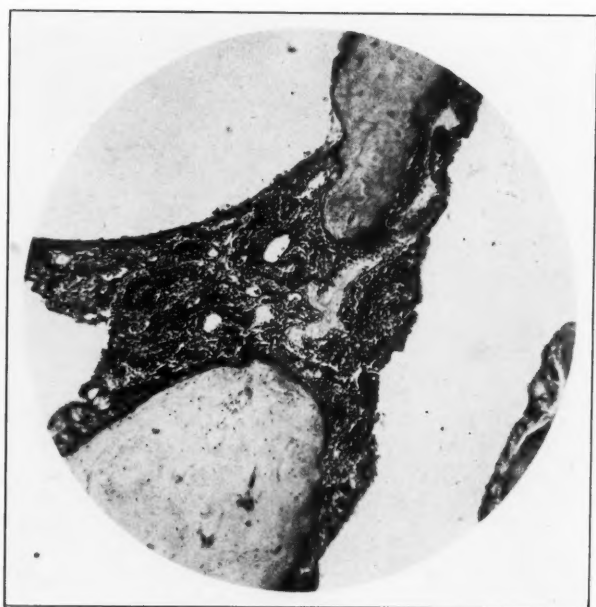


Fig. 15. Site of round window, rabbit No. 4. High power. Continuity of inflammatory tissue of middle and internal ear cavities through remains of membrane.



Fig. 16. High power view of a whorl of the cochlea in rabbit No. 4. Shows severe destructive changes and spread of inflammation down the modiolus by perineural lymphatics.



Fig. 17. Internal acoustic meatus, rabbit No. 4. Shows carious bone surrounded by inflammatory reaction. Macula in same field shows practically no change.



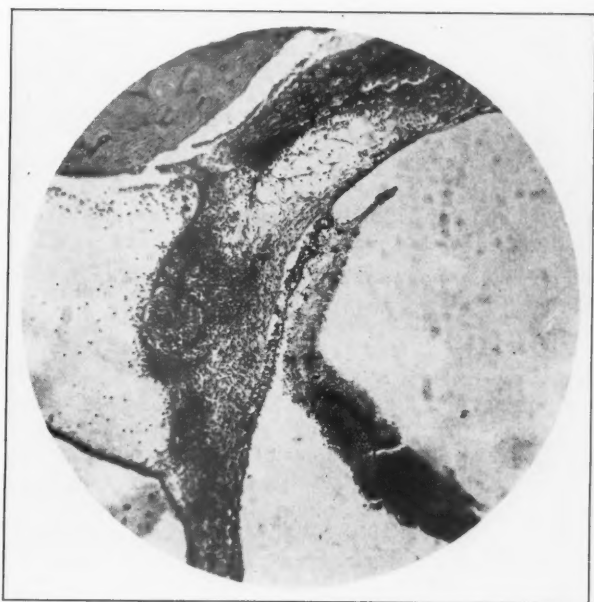


Fig. 18. Submucous inflammatory reaction under macula sacculi of rabbit No. 4. The otolithic membrane has been partially displaced from its normal contact with the hair cells.



LXII.

SOURCES OF ERROR IN SINUS RADIOGRAPHY  
WITH FLUID CONTRAST MEDIA.\*

BY ARTHUR W. PROETZ, M. D.,

ST. LOUIS.

At the present time the opaque filling of nasal sinuses for radiographic study serves a twofold purpose. It defines the exact limits of the cavity; and it furnishes accurate information regarding the drainage of the cell. That it serves these purposes admirably is now generally admitted, but too much cannot be said of the importance of special familiarity with the requirements of the method before diagnosis is attempted. Failures in diagnosis can usually be traced to some unfamiliarity with the mechanics of fluids, or to failure to observe the few simple rules of radiographic technic.

Regarding the topography of the cell, the use of contrast media has but one function, namely, exact definition of the cell cavity, for the purpose of determining its form and of comparing it with the bone cavity. Any mechanical factors which may cause the oil shadow to simulate any other condition than that which actually exists, vitiate the results of the investigation. Shadow pictures are notoriously deceptive. The silhouette of the rabbit on the nursery wall gives little information regarding the nature of the hands which cast it. It is obvious that the projection of a three dimensional body upon a two dimensional plane must open avenues for error.

Simple filling accentuates any discrepancies which may exist between the cavity and the bony wall. These discrepancies are called "filling defects" and may be anything from a slightly thickened membrane to complete obliteration of the cavity. Any of these may be simulated by shadows, arising through faulty technic, of other conditions than those suspected, as will be shown.

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\*Read before the Colorado Congress of Ophthalmology and Otolaryngology, Denver, Colo., July 21, 1928.

If one allow his observations to end with the topography of the cell, he ignores at least half the diagnostic information laid before him. The behavior of oil as it enters the ostia of cells (by displacement) and as it is spontaneously drained from them (after introduction by any method without astringents) is a reliable index to the drainage of the cell, and drainage is the objective of most sinus surgery.

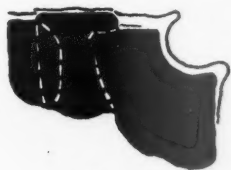


Fig. 1.

the sphenoid. (Fig. 1.) Bilateral filling demands constant comparison between postero-anterior and lateral films in order to identify individual cell shadows. Radiography with the horizontal beam minimizes this error.

The projection of a group of overlapping cells, such as ethmoids and sphenoids in two dimensions, naturally demands some care in singling them out. The anatomical irregularity of individual cells gives rise to irregular shadows, even though only a single cell is filled. For this reason it is sometimes difficult to determine whether the posterior ethmoid cell is filled with the sphenoid or not. Posterior ethmoids sometimes overlie a sphenoid to such extent that they are injected—in fact, sometimes surgically opened—under the impression that the sphenoid has been dealt with. If by good fortune there is any pneumatization of the pterygoid process in such a doubtful case, a lateral film made with a horizontal beam will at once clear up the difficulty as the extension naturally indicates the sphenoid. (These pneumatized pterygoids are not rare. In my own series, they are of the order of 20 per cent.) Cell identity can be learned only by observing a large number of films, and in this connection it is exceedingly important to maintain exactly the same angulation throughout the series. For example, the pneumatized pterygoid process of a sphenoid cell just mentioned, might easily be mistaken for a high maxil-

lary filling in a postero-anterior film. The lateral film should dispel any doubt as to its identity.

Particularly difficult of interpretation is that situation in which the sphenoid of one side is small and anteriorly situated in the bone, while its fellow is large and lies not only beside but behind it as well. Such a small sphenoid may readily be mistaken for a posterior ethmoid cell and, so far as the cell itself is concerned, its identification has little clinical significance, because it is accessible, no matter what it is. It is important from a surgical standpoint, however, to know whether the space behind it communicates with the opposite side, which it will do if it is an arm of the opposite cell, but which it will not do if it is a sphenoid covered by what turns out to be a posteriorly placed posterior ethmoid. (Fig. 2.)

Absolutely essential to the correct interpretation of a film is the knowledge of the relation of tube, head and film in making the exposure. Shadows of the same oil-filled cell, in different positions, vary so widely that, in my experience, a film is utterly worthless unless these data are known.

Two technical questions still occupy the attention of radiologists: First, shall the sinus be partially or completely filled? and second, what is the optimum tube position?

There are competent clinicians who maintain that complete filling is essential to complete diagnosis. In my own work, I have adopted the partial filling in all cases for two reasons. To begin with, complete filling by any method almost never occurs. If the oil is introduced through the normal opening or through a puncture wound, the filling can proceed only until the oil reaches the ostium, when it overflows, leaving a bubble of air above that level, which no amount of manipulation will

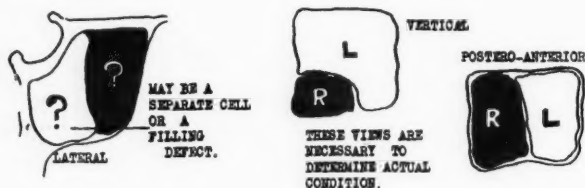


Fig. 2.

dislodge. (Fig. 3.) If a vertical septum extends downward from the top of a cell, it forms a cul-de-sac in which air will also remain after filling. (Fig. 4.) There is only one physical situation in which perfect filling can be accomplished and that is in a cell without septa, having its ostium exactly at the extreme top. (Fig. 5.) Such a situation must necessarily be rare, and since there is no way of knowing in what position of the head the ostium will be exactly at the top, one can never be certain of complete filling. Throw an empty bottle into the water; introduce a cannula connected to a syringe and try to fill it by squirting water into it. So long as the position of the ostium is out of control, the bottle cannot be filled.



Fig. 3.

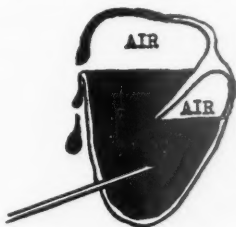


Fig. 4.

the opening of the tube, not because this is the simplest method but because it affords information which the vertical view from above does not. It tells us something about the shape of the tube, and the density of the fluid and reveals the presence or absence of a precipitate. We know that the fluid is everywhere in perfect apposition to the glass, being held there by gravity and that, should a translucent globule be discovered anywhere beneath the surface of the fluid, it cannot possibly be an air bubble. If the

Not only for this reason, have I resorted to deliberate partial filling but also because, by its use with a horizontal ray, the definite horizontal oil levels are made to supply additional information.

I try to examine an oil-filled sinus much as one would examine fluid in any other container. One naturally examines the fluid in a test tube from the side, instead of looking down into



Fig. 5.

oil shadow is to tell us anything regarding the cell contour, this accurate contact is obviously necessary. Since fluid seeks its lowest level, observation from above often reveals the shadow of the margin of the oil pool, which in itself has no

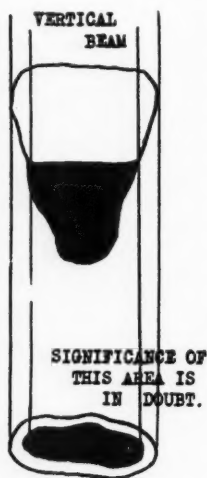


Fig. 6.

significance but which closely simulates the picture of incomplete filling, due to a thickened membrane and may be confounded with it. (Fig. 6.) In the case of overlapping cells, when X-rayed from above, a conglomerate shadow results, in which it is almost impossible to distinguish shadows of individual cells. If the same cells are rayed from the side, with a horizontal beam, against a vertical film, the shadow is still conglomerate, but there will be as many straight horizontal oil level shadows distinguishable as there are cells, greatly facilitating the sinus-mapping. In the case of thickened membranes and other filling defects caused by inflammatory conditions, a satisfactory silhouette is obtained of the floor of the cell where such defects are most common. (Fig. 12.)

In connection with polyps, it should be borne in mind that the tip of the penetrating trocar may enter the polyp and deposit a certain amount of oil within it while the true cavity remains empty. The resulting radiograph will naturally be misleading. (Fig. 7.)

Washing the sinuses immediately before introducing iodized oil, even though followed by an air current, is not to be recommended. Water remaining in the cell alters the oil shadow by causing the oil to assume globular shapes instead of adapting itself accurately to the cell wall. If any secre-

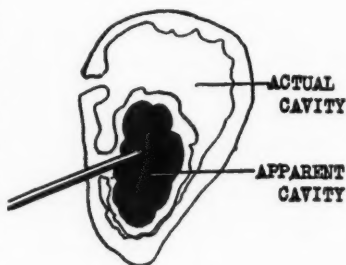


Fig. 7.

tion which may be present is sufficiently fluid, it will float on the top of the oil, owing to the high specific gravity of the latter. If it remains at the bottom it will show in the radiograph as a translucent mass and inform us that an abnormal condition is present. If it is desired to eliminate a known exudate before oil injection, for the purpose of determining the exact wall contour, then the washing should be done twenty-four hours before lipiodol is introduced and the patient instructed to lie so as to facilitate drainage.

When a cell is only partially filled, slight variation in the head position causes pronounced variation in the shadow of the oil pool as it adapts itself to the dependent portion of the sinus. Shadows with the vertical beam will therefore vary widely with the shape of the pool. A small amount of oil confined to a depression, produces a small shadow, whereas the same amount of oil spread out on a neighboring plateau by a slight change in the head position, may cause the sinus to appear almost completely filled. (Fig. 8.) As the horizontal-beam-shadow silhouettes the depth of the pool instead of its surface, the variations are small and the relations of oil-shadow to bone-shadow are much more likely to approximate the true condition. (Fig. 9.)

Aside from the topography of the cells, contrast media have, in my experience, yielded their best results by demonstrating with the clarity of diagrams, the drainage facilities of the various cells. This study of relative drainage-mapping has proved far more valuable from the clinical standpoint than the mere topographical information, in determining which sinuses to open and which to let alone. The cells are filled by displacement without the use of astringents and a radiograph is made. Subsequent films, made at definite intervals, graphically demonstrate the facility with which each individual cell is spontaneously drained. One precaution is of paramount importance: The successive films should be made at exactly the same angle as the initial film, for satisfactory comparison.

It is very rare that an individual sinus proves to be infected while its neighbors remain normal; in other words, nasal examination often indicates infection in whole groups of con-

VERTICAL BEAM  
SAME CELL...SAME FILLING...DIFFERENT ANGLES

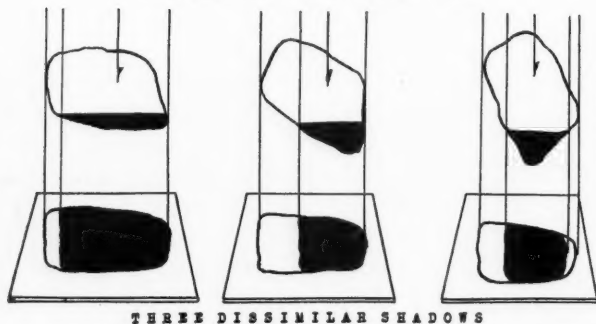


Fig. 8.

HORIZONTAL BEAM  
SAME CELL, FILLING AND ANGLES AS IN  
THE PREVIOUS FIGURE.

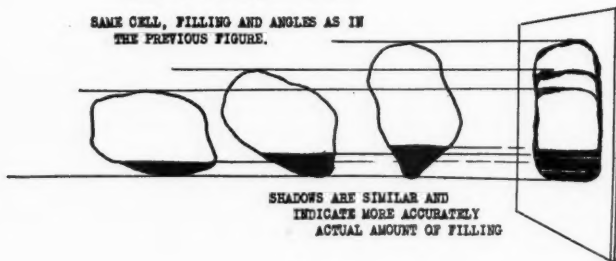


Fig. 9.



tiguous cells. More often than not, an individual member of the group supplies the derogatory influence which maintains the entire infection. This is usually due to some obstruction to drainage and aeration, the eradication of which clears up the whole infection.

If lipiodol is introduced into the entire group of cells by displacement, the offending cell fails to fill, provided that its ostium is sufficiently obstructed or that it is already filled with some other fluid—namely, an exudate. (Fig. 10.) This failure to fill rarely occurs, as obstruction is rarely complete. If, however, the entire group is filled and allowed to empty itself spontaneously, the retained oil in the diseased cell will stand out as a prominent shadow. (Fig. 11.) It is thus often possible to select individual diseased cells from a group for treatment. This emptying may be observed by making a film as soon as the sinuses are filled and repeating at twenty-four hour intervals. In order to save time and expense, I have made it a practice to make only the routine set of first films and one seventy-two hour film (usually lateral, unless the first films show the postero-anterior position to be more informative).

One of the requisites in the careful estimation of this drainage is that all the exposures shall be made with tube, head and plate in exactly similar positions—in fact, it is advisable to maintain great exactitude of routine angulation in all lipiodol work of the skull. Familiarity with the shadow details of each position makes for much higher efficiency in interpretation. Toward this end, Ernst has devised a precision apparatus, presented before the Second International Radiologic Congress in Stockholm (1928), for use in sinus work, by which the beam focuses on the center of the film, no matter what angle the tube assumes in relation to the head. The patient is set in position and the base line (outer canthus to external auditory meatus) is determined. This is set at right angles to the film, and the tube is then accurately adjusted to any desired angle above or below the line. By this proceeding, standard shadows of the head are obtained, and variations from the normal spring into view, which would otherwise be overlooked. This simple apparatus has proven of inestimable value in the identification of vague shadows.



Fig. 10.

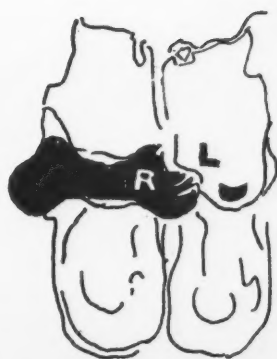
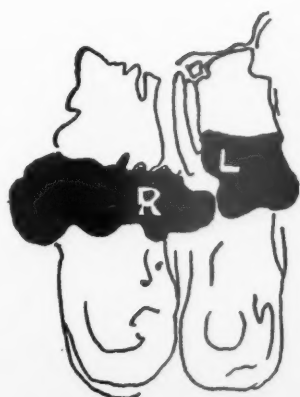


Fig. 11.





Fig. 12-A.

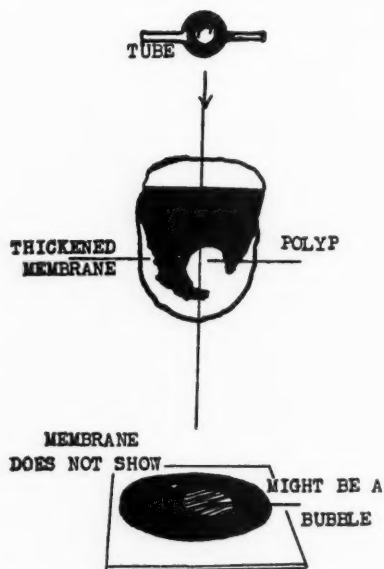


Fig. 12.

## SUMMARY.

1. Most errors in sinus diagnosis by means of fluid contrast media are the result of unfamiliarity or inexperience with the method and are not due to any shortcomings of the method itself.

2. Certain simple precautions should be followed, such as are observed in inspecting fluids in any other translucent containers.

3. Partial filling and the horizontal beam have proven most satisfactory in my hands.

4. Adventitious causes of error should be avoided, such as washing immediately before oil injection.

5. Standard positions should be accurately adhered to in order to familiarize oneself thoroughly with the contours of the complex sinus-shadows. Precision apparatus has been designed for this purpose.

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### LXIII.

#### ABSCESS OF THE BRAIN OF OTITIC ORIGIN.

BY JOSEPH H. BRYAN, M. D.,

WASHINGTON.

The following is a report of an interesting case of abscess of the brain following a long period of middle ear disease.

McKeough, B. J., male, white, age 23, student. June 1, 1926, patient admitted to hospital complaining of severe pain in left ear which had to be controlled with opiates. Temperature 101. Marked vertigo.

Family history: Entirely negative.

Past history: Grossly negative. Patient was born in Wisconsin and has lived there practically all his life. Had usual diseases of childhood. Has had some trouble which he called chronic inflammation of left ear, since eight years of age. Patient has been studying exceptionally hard during the school term just past and was a candidate for his M. A. degree.

Present illness: Present illness began about May 1st, when patient was taken with a severe pain in the left ear. Discharge, which had been slight for about two a half years, became profuse. He sought treatment for the condition, which gradually became worse, marked by severe pain in and about the left ear, discharge and some vertigo. This was the condition in which patient was found when he was admitted to hospital.

#### PHYSICAL EXAMINATION.

General: Patient is a young white male, fairly well developed, lying in the dorsal decubitus position, complaining of great pain in left ear and dizziness, more marked when disturbed.

Head, neck and eyes negative. No muscular weakness. No nystagmus, no ptosis of lids. Pupils moderately contracted and react to light and accommodation equally. Fundus entirely negative, no evidence of intracranial pressure.

Nose and throat: Mucous membrane congested. Tonsils inflamed.

Ears: Right ear negative, no discharge, no tenderness. Left ear draining profusely, no tenderness over mastoid. Canal negative. Tympanic membrane eroded by chronic inflammatory condition. Polyp emerges from middle ear.

Neck: Palpable glands present. No tenderness. No muscular rigidity.

Chest: Lungs negative. No abnormal dullness. No adventitious sounds.

Heart: Not enlarged. Rate moderately increased. Sounds of fairly good quality. No murmurs or arrhythmia.

Abdomen: Negative. No tremors, no rigidity, no tenderness over epigastrium, gall bladder or appendix. Liver and spleen not palpable.

Extremities: Negative. No paralysis or weakness. No rigidity or spasms. No Kernig.

Reflexes: Pupillary present and active.

Triceps, biceps, knee jerk: Present but slightly sluggish.

No abnormal reflexes. No ankle or knee clonus. No Babinski.

Mental examination: Mentality somewhat sluggish. Patient appears drowsy; speech is slow but rational.

June 2, 1926. Patient still complaining of severe pain on left side of head. Vomited twice but not projectile (may be due to codein). Patient still dizzy. Temperature reached 103.6, which was followed by severe chill of short duration.

Spinal puncture: (a) Slight increase in pressure; (b) six cells per cm.; (c) 50 per cent polys, 50 per cent lymphocytes; (d) marked increase in globulin; (e) culture negative.

Urine shows granular casts and trace of albumen.

Leucocyte count, 11,650. X-ray of mastoid, complete obliteration of all cellular structure on left side. Bone density considerably increased. Chronic disease of left mastoid.

June 3, 1926. Not much change in condition. Short chill. Temperature showed wide excursions from 99.6 to 103. It reached 104.6 at one time. Patient transferred from ward to private room. Still in great pain, still dizzy. No eye symptoms, no evidence of paralysis or rigidity of muscles.

June 4, 1926. Temperature 103 (axilla), pulse 100, respiration 22. Chill lasting 25 minutes; several shorter chills during 24 hours. Nausea and vomiting. Perspiring freely from time to time. Pain in area of left ear persists.



June 5, 1926.—Temperature reached 104 (axilla); septic in type. Pulse good. Patient irrational at times. Has some motor aphasia (difficulty in explaining himself). Frequent chilly feeling. Severe headache radiating from left ear.

June 6, 1926. No change in condition other than gastrointestinal symptoms which developed with gas and some abdominal rigidity.

June 7, 1926. Temperature 102, pulse 88, respiration 22. Pain developed in posterior cervical region with some slight rigidity of these muscles. Slightly irrational at times.

Spinal puncture: (a) Turbid fluid; (b) moderate increase in pressure; (c) 555 cells per cm.; (d) 90 per cent polys, 10 per cent lymphocytes; (e) marked increase in protein; (f) culture negative. Somewhat more comfortable following spinal puncture.

June 8, 1926. General condition much worse. Mental condition worse. Temperature between 102 and 104 with small excursions.

Neurologic examination by Dr. D. V. Stuart: (a) There was mental hebetude with some motor aphasia, failure of recognition of objects when handled, and failure of recognition of words heard.

(b) All deep reflexes were hyperactive in both upper and lower extremities. This was more marked on the right side.

(c) Slight retraction of head and rigidity of the posterior muscles.

The ocular fundi were normal in appearance, and there was no disturbance of function of any of the cranial nerves so far as could be determined. Muscular strength in the trunk and extremities was equal on the two sides and apparently unimpaired. Because of the patient's condition neurologic examination was impossible.

Impression: Brain abscess, left temporal lobe, and diffuse meningitis.

On account of the past history of the case, it would seem that the abscess was of long standing. Meningitis, on the other hand, must have been of recent origin, as evidenced by the different spinal fluid findings one week prior to the time I saw Mr. McKeough, and again twenty-four hours later.

Patient operated upon under local anesthetic, Dr. H. H. Kerr operating. Opening in skull in left temporal region; dura incised and abscess with free pus opened and drained with tube. Abscess in lower portion of temporal lobe.

After operation temperature reached above 102 but once. Urine shows trace of albumin.

June 9, 1926. Condition slightly improved, more rational. Had fifteen-minute chill.

June 10, 1926. Patient much worse. Bladder and rectal reflexes abolished.

June 11, 1926. Condition gradually getting worse. Abdomen rigid and cramp-like pains present. Rigidity of posterior cervical muscles. Irrational. Temperature and pulse the same; respiration labored.

Spinal puncture:

(a) Fluid cloudy, under no pressure.

(b) 3050 cells per cm., 89 per cent polys.; 11 per cent lymphocytes.

(c) Marked globulin increase.

(d) Culture shows influenza bacillus.

Culture from brain negative. Blood transfusion, 450 cc. Restless, pulling covers off, irrational.

June 12, 1926. Condition still worse. No rest. Another transfusion 500 cc. Respirations very labored and rapid.

Respiration ceased at 10:15 p. m.

#### AUTOPSY.

Name: B. J. McKeough; age 23; color, white; student.

Date of autopsy: June 12, 11:45 p. m.

External appearance: The body is that of a large, well developed, but greatly emaciated white man of about 23 years of age. There is a trephine opening in the left temporal region from which protrudes a drainage tube. Drainage through this tube consists of a thin, limpid, light brown colored fluid with very offensive odor. There are no other external marks of any importance.

Head: On removing the calvarium the dura, except for moderate amount of injection of the vessels and the opening through which the drainage tube passes, is grossly normal. Upon removing the drainage tube a cavity is seen in

the brain substance; there is no evidence of an attempt of the brain to herniate through this opening in the dura. On removing the dura covering the left cerebral hemisphere there are seen areas of yellowish fibrinous exudate scattered at various points, particularly just to the left of the medium line and toward the frontal lobe. There is also an escape of semipurulent cerebrospinal fluid. The hole made by the drainage tube seemed to be well forward and into the lateral surface of the left temporal lobe. Pressure in this region produces an outpouring of slightly thick, greenish yellow pus, some old blood and much débris, all of which has a very offensive odor. The dura over the right cerebral hemisphere is intensely congested as are vessels in the cortex on this side, although there are no deposits of the fibrinous exudate seen on the opposite side. The entire temporal lobe and the fossa in which it lies is lined and covered by the same type of exudate noted above. Beneath the tentorium cerebella on each side is a deposit of greenish yellow pus. The entire base of the brain, well down into the spinal canal, is bathed in this material. Beneath the dura in the left temporal fossa, extending from the petrous portion of the temporal bone, is a collection of pus. There is no superficial destruction of the petrous portion of the temporal bone or in either the anterior or posterior fossa of the base. The internal auditory meatus is filled with pus, necrotic material and old blood. The lamina cribrosa is destroyed and all the structures of the internal and middle ear are apparently necrosed as evidenced by the free passage of a large size probe from the external to the internal auditory meatus as well as by the amount and character of material of extremely offensive odor escaping from both orifices.

Thorax: The muscle and subcutaneous tissues covering the thoracic cavity is of an intense chocolate brown color. The right pleural cavity is normal except for a few adhesions at the apex. The left pleural cavity is normal except for a small amount of clear, straw colored fluid. Both lungs show a moderate amount of hypostatic congestion. The pericardium contains an excessive amount of clear, yellow colored fluid. The heart is in systole. The musculature is firm and well developed. The chambers are empty. There is no gross pathology in the myocardium or endocardium.

Abdomen: There is marked distension of the stomach, small intestine and particularly the large intestine. The appendix is of the retrocecal variety and is bound down by a number of adhesions. The spleen is large, soft and shows typical appearances of an acute infection. The liver is grossly normal. The other abdominal organs not examined.

Anatomic diagnosis: (1) Acute purulent meningitis; (2) brain abscess; (3) destruction of the internal and middle ear of the left side, bone necrosis.

Section of the brain shows an abscess cavity about the size of a dime in circumference and about one and one-half centimeters in diameter lying in the extreme posterior portion of the temporal lobe and which comes to the surface of the os with the lateral ventricle. The lateral ventricle is filled with pus, débris and necrotic tissue, some of which necrotic areas extend beyond the normal ventricular limits. A second abscess cavity into which the drainage tube extended and which also communicated into the ventricle is well forward on the lateral surface and above the junction of the temporal and parietal lobe.

Figures No. 1 and No. 2 show abscess in the cerebrum and in the lateral ventricle.

Abscess of the brain is a subject most interesting to us as otologists, and I believe all carefully observed cases should be recorded and freely discussed, for it is only in this way that we can expect to reach a uniform method of localization, and adopt the proper surgical measures for the relief of the abscess.

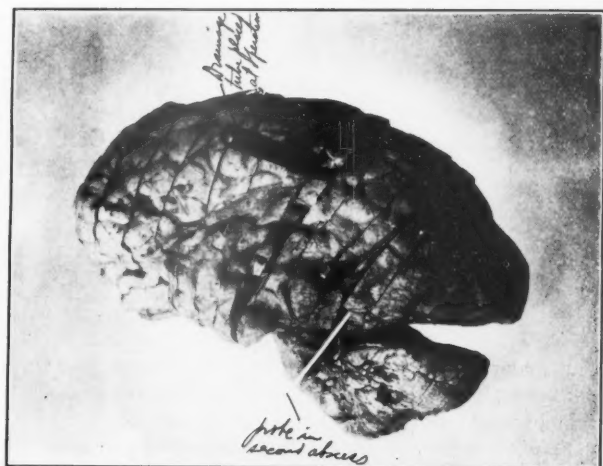


Fig. 1.

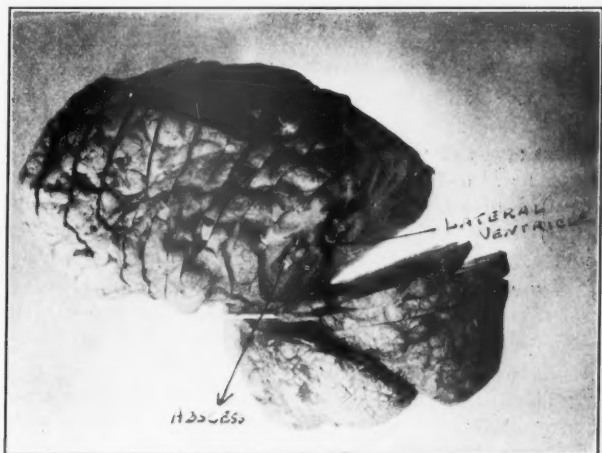


Fig. 2.

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#### LXIV.

### FRACTURE OF THE MALAR ZYGOMATIC ARCH— REVIEW OF THE LITERATURE—A SIMPLIFIED OPERATIVE TECHNIC—CASE REPORTS.\*

BY SAM E. ROBERTS, M. D.,

KANSAS CITY.

#### ANATOMY.

The malar bone underlies the most prominent part of the cheek, commonly called the cheekbone. It forms the outer and lower border of the orbit. The bone is convex from side to side. The most elevated part of the convex outer surface forms the malar tuberosity. It articulates with the frontal, sphenoid, superior maxillary and zygomatic process of the temporal bone.

In this age of rapid transportation by automobile and aeroplane certain fractures about the face and head, which were rare in the past, are now becoming much more common. For this reason, it seems apropos to review this subject and bring forward any additional information.

Each age of mechanical progress seems to come hand in hand in with additional hazards. For example, the high construction of the cowl of the training ship used on the American aviation fields during the World War was the cause of injury and produced a mark on the bridge of the flier's nose almost as characteristic as the scars from fencing on the face of the Heidelberg student.

Fracture of the malar has been rare in the past, if one is to judge from the literature on the subject. Ten standard textbooks of surgery and fractures<sup>1</sup> do not even mention it.

About two years ago I was suddenly confronted with a fracture of the malar and, with a hurried search of the literature I found no help whatsoever. Fracture of the zygomatic portion of the arch is so often associated with fracture of the malar

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\*Accepted as candidate's thesis for Fellowship in American Laryngological, Rhinological and Otological Society, Inc.

that it is difficult to separate them in this discussion or in the review of literature to follow.

#### REVIEW OF THE LITERATURE.

Duverney,<sup>2</sup> in 1751, was the first to mention this fracture. He reported two cases: one a French soldier, the other an infant, 3 years old. He was supposed to have reduced the fracture by pressure from within the mouth. There seems to be a lapse in the literature of 106 years, the next report being by Hamilton in 1857.

Hamilton<sup>3</sup> quotes Duverney, and says: "Examination of the interior of the buccal cavity will convince one that it is impossible to correct by Duverney's method when the fracture is at any point near the middle of the zygoma, and that it is only possible when the fracture is at or near the junction of the zygoma with the body of the malar bone."

Kemper<sup>4</sup>, in 1922, stated there were less than one dozen cases of fracture of the zygomatic arch in the catalog of the Surgeon General's library. This comprised the medical literature of the entire world up to that time. I have been able to find about fifteen additional reports. Kemper tells of one case in which he claims to have reduced the fracture by using his thumb in the buccal cavity, crowding the bone upward and into place. He also stated that distinguished surgeons asserted the zygomatic malar arch could not be reached through the buccal cavity. I am inclined to agree with the latter.

Many of the reports do not state whether the fracture occurred in the zygomatic process alone or in combination with the malar; in fact, the malar is seldom mentioned. It can also be seen there are many different methods of reducing the fracture, and the literature is quite confusing and contradictory.

Bellamy<sup>5</sup> reports one case in which an unsuccessful attempt was made at reduction through the buccal cavity, with the thumb pushing upwards on the bone. He states that as soon as the masseter muscle was in action it drew the fragments of the bone down into its former position.

Matas,<sup>6</sup> in 1896, described a zygomatic fracture. His classical method of treatment still stands, in my opinion, as the best *modus operandi*. His method of reduction was as follows:

"A long, full curved (semicircular) Hagedorne needle, threaded with silk as a carrier, was made to penetrate the skin about one inch above the midpoint of the displaced fragment and was carried well into the temple fossa under the broken bone. Then the point of the needle was raised and made to emerge about one-half inch below the lower border of the broken arch. As the needle was pulled out, a strong silver wire, about one foot long, was attached to the silk carrier and dragged through the tract of the needle, so as to form a metallic loop under the misplaced bone. By twisting the ends of the wire together, a loop was formed which permitted strong and easy traction to be made on the broken fragment. With the index and middle fingers hooked under the wire, loop traction was begun by pulling directly upward and outward. The moment this traction began, the displaced fragment yielded and instantly returned to its normal position with a snap that was loud enough to be heard by the gentlemen who were assisting." Dr. Matas also advises a splinting with an ordinary glass slide placed parallel with the long axis, the anterior end resting on the malar prominence and the posterior on the root of the zygoma. The wire was twisted firmly over the slide.

Gibson<sup>7</sup> reported one case reduced by open incision.

Manwaring<sup>8</sup> describes the use of the "cow-horn" forceps used by dentists in reduction of the fracture of the zygomatic portion. One point is inserted above and one below, penetrating the skin and passing underneath the depressed bone. By raising the forceps upward the depression is corrected. He states this may also apply to fractures of the malar.

Morehead<sup>9</sup> states the fracture cannot be reduced without exposure through the cheek or mouth. "Traction" by hook or some other device is employed. If the zygoma alone is involved he states: "Pressure is usually effective applied outside and inside the cheek, as by a pair of curved forceps, one blade of which rests on the zygoma and the other within the mouth against the upper teeth."

Bower<sup>11</sup> reports one case of fracture of the zygomatic arch with treatment by incision just above the zygoma. Depression was raised with an ordinary elevator.



Ellis<sup>12</sup> reports treating a case by incision and hook behind the depressed arch.

Keen<sup>13</sup> states there are three methods of operating: "1. Direct incision is made through the skin and fragments pulled outward. This is objectionable on account of the scar and inability to retain the fragments in place. 2. Through the mouth by inserting blunt instruments beneath the bone from within and lifting the fragments up. 3. Method of Lothrop—he recommends making a small incision through the mucous membrane of the mouth just under the canine fossa and through an opening in the anterior wall of the antrum and lift up the fragments, packing the antrum with gauze, which is not removed for four or five days." He adds: "This would seem like an ideal method were it not for the danger of infection."

Lloyd<sup>14</sup> reported one patient treated by open incision directly across the arch. He does not state how the bone was elevated.

Clark<sup>15</sup> reported one patient in which no treatment was given and he states Nature accomplished the cure.

Williams<sup>16</sup> reports one patient in which apparently the Matas technic was used unsuccessfully. He resorted to open incision.

Lehman<sup>17</sup> reports two cases. Both were treated by incision over the arch, and reduction was accomplished by a "lifter." He also states: "Reduction from the buccal cavity is not possible, since the temporal muscle blocks the entrance."

Treves<sup>18</sup> states: "In case of fracture with depression of the zygomatic arch, no treatment is necessary if the lower jaw can be used without any trouble. If the movement of the jaws is interfered with an open incision is made and the depressed fragment elevated and kept in position by wiring, if necessary." He reported one case in which Matas technic was followed.

Todd<sup>19</sup> performed experimental work with skulls—373 Europeans and 94 negroes. He states that the depression is usually more pronounced when the fracture occurs at the zygomatic malar suture. It may involve the malar, zygoma or both, but the temporal mandibular joint is never involved.

Duchange<sup>20</sup> says: "Treatment is either easy or impossible. A fractured malar bone and zygoma cannot be treated after several days of delay. They must be treated as soon as diag-

nosed." His method of reduction was to push on the bone from below.

O'Brian<sup>21</sup> reported two cases, the method of treatment not stated.

Roberts and Kelly<sup>22</sup> advised an open operation and direct elevation. No cases reported.

Malgaigne<sup>23</sup> reports that although he has never met with this type of fracture, he is able to collect only five cases.

Hamilton, H.,<sup>24</sup> reports one case of untreated fracture of the zygomatic portion of the arch. The patient was unable to open or close his jaws. The case was untreated. At the end of five weeks the mobility was nearly but not quite restored. The external depression remained.

Ranking<sup>25</sup> reports one case but does not state the treatment given.

Weir<sup>26</sup> reports two cases of malar fracture. The first case was treated by incision through the mucous membrane, just above and outside the right upper canine tooth. The antrum was opened and a steel sound was passed upward and the depressed bone was loosened and raised into proper place. The antrum was packed. The result was not stated.

Hiffelsheim<sup>27</sup> reports no treatment.

Leger<sup>28</sup> reports one case but no treatment.

Garner<sup>29</sup> reports a case of depressed fracture of the malar bone and zygomatic arch relieved by open operation through the mouth.

#### SYMPTOMS.

The external swelling around the orbit of the zygoma usually obscures the signs of bone depression. Where the malar is involved extending into the orbit the depression can be felt, even in the presence of the swelling, by starting at the inner canthus and running the finger along the lower orbital margin. A distinct break and depression will be noticed, extending, as a rule, to the external canthus. Where the involvement is more in the zygoma the depression is more marked the nearer one approaches the external auditory meatus; the movement of the jaws is difficult because of injury to the masseter muscle lying directly underneath.

## A SIMPLIFIED TECHNIC FOR REDUCTION OF MALAR FRACTURES.

A general anesthetic should always be used. The whole side of the face is prepared with the same precautions as though an open operation were to be done.

A stab is made with a sharp pointed knife, about three-fourths of an inch directly below the external canthus. (Illustration No. 1.) This puncture is carried down into the bone through the periosteum. (Illustration No. 2.) As a rule, only a small amount of bleeding occurs. The screw (Illustration No. 3) is then inserted (as shown in Illustration No. 4), with a half turned motion in a perpendicular plane with the flat surface of the bone. Considerable pressure may be necessary in older patients to penetrate the bone, and the depression from the fracture may be exaggerated at this stage. This, however, is of no consequence, for as soon as one full turn of the screw has passed through the flat bone it may be elevated to normal position. The bone can be felt to "give" as the lift upward is made, and there is little danger of overcorrection if done firmly and slowly.

With the screw still in place, one may feel along the lower bony floor of the orbit and will find the normal contour restored. The screw is then removed from the bone and the stab wound closed with one silk thread stitch and a collodion dressing applied. The stitch is removed in 48 hours to prevent marking. After one or two months one must look very carefully to see any trace of the procedure.

There are three distinct advantages of this operation:

1. The simplicity.
2. The operative field may be rendered aseptic, eliminating possibility of infection as might occur if any procedure were attempted through the mouth or maxillary antrum.
3. No remaining scar on the face, as would occur after any open procedure in this region.

The operation should be performed as soon as possible after the accident, before repair has begun and fixation, in a false position, has occurred. I have not, as yet, tried this procedure on old fractures and am unable to state whether or not it would be efficacious. Of the three cases I have seen, the oldest fracture was three weeks and was by far the most difficult to

reduce. The first was less than 24 hours after the accident and was the most easily reduced.

#### CASE REPORTS.

1.—A boy, age 17, son of a physician, while engaged in a football contest, was tackled and thrown forward, striking on the left side of his face. There was considerable shock and play was discontinued. A marked depression of the left malar region was immediately observed by his father. Examination, two hours later, showed a depression extending from slightly posterior to the malar tuberosity forward to include about one-half the lower orbital floor. The patient was immediately admitted to the hospital. The following morning he was given a general anesthetic and the bone was elevated into normal position by the technic previously described. The results were perfect and the scar practically invisible.

2. A man, aged 47, while intoxicated, was either struck with an opponent's fist or fell forward on his face. The physical findings were practically the same as in Case No. 1, and the treatment was identical. The fracture occurred seven days before the operation. More difficulty was experienced in elevating the bone, but the result was equally as good.

3. A woman, age 45, three weeks previous to hospital admission, was in an automobile accident. She had a depression of the right malar and zygoma, partial paralysis of the right facial nerve, immobility of the jaws, impaired hearing on the right and a great deal of pain over the fifth nerve region.

I could not account for all these findings without some central nervous system lesion. The neurologist, however, reported negative findings. Three days later, under a general anesthesia, with considerable difficulty, I elevated the malar. At that time I assumed the zygoma was also elevated. The mobility of the jaws did not return for six weeks, and the malar deformity was corrected only in part, according to reports. The patient resides in Texas, and I have been unable to observe her since she left the hospital. I do not consider, however, my result entirely satisfactory. I should have used the Matas technic and elevated the zygoma after elevating the malar.

NOTE.—Since the above was written I have had a chance to see this patient and demonstrate her case before the Kansas

City Eye, Ear, Nose and Throat Society. The result proved to be excellent. There was no remaining depression either in the malar or orbital region.

1810 FEDERAL RESERVE BANK BLDG.

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I. Cross (X) indicates site of stab incision.



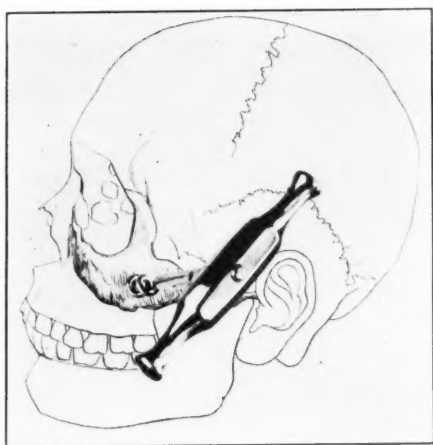


II. Cross (X) marks point where bone is entered.





III. Screw made by Mueller for penetrating malar bone.



IV. Screw entering malar bone.

## LXV.

### FURTHER DEVELOPMENT OF THE TRANSSPHENOID APPROACH TO THE OPTIC FORAMEN.

BY EDWARD CECIL SEWALL, M. D.,

SAN FRANCISCO.

In 1926 I published some anatomic studies on this subject, in connection with technic developed for the external operation under local anesthesia, on the frontal-ethmosphenoid group of sinuses. In operating in this manner I frequently approached so closely to the optic foramen that the removal of its walls suggested no great difficulty from a technical standpoint. I worked out the anatomic details on the cadaver and presented some operative possibilities before the meeting of the American Laryngological Society in 1926. Since this time my interest in this subject has increased. I have operated upon one patient according to the then described method, and I have worked out further details and have widened the scope of the operative technic. In presenting these anatomic studies before this joint meeting of the Triological and the Pacific Coast Otolaryngological societies, I appreciate the benefit of criticism from both the rhinologic and ophthalmologic point of view.

There are a number of pathologic conditions for which such an operation as herein outlined might be useful. I mention optic neuritis, optic tract tumors and constriction of the nerve by the optic canal or by fracture through its walls. All offer serious conditions, difficult of relief. Other pathology in the immediate neighborhood may also be found amenable to this approach. The difficulties of the operative methods now in vogue may be partly responsible for the present hesitancy in operating. Van der Hoeve<sup>1</sup> says that "The canal operation for the preservation of visual power should be performed only if both optic nerves are endangered in such a measure that absolute blindness is to be feared." Three routes of approach to the optic canal have been employed. Schloffer<sup>2</sup> used a flap of the skin, periosteum and bone of the upper orbital wall. He raised the frontal lobe of the brain and approached the optic canal

from in front. Hildebrand<sup>3</sup> entered the orbit under the brow, chiseling off a bit of the orbital edge. He retracted the eye downward, pierced the orbital roof near the optic canal and with thin forceps enlarged the optic foramen. Dandy<sup>4</sup> entered by the temporal route and approached the foramen by raising the frontal lobe of the brain. It is conceded that such approach may be necessary for certain intracranial conditions.

Of the various pathologic conditions for which such an operation is suitable, optic neuritis, because of its frequency and gravity, is of most importance. Optic neuritis, papilledema, choked disc, papillitis and neuroretinitis are terms in common usage to express pathologic changes that have taken place in the optic nerve or optic disc. Papilledema and choked disc seem best used to designate the edematous changes in the nerve head resulting from intracranial pressure. Optic neuritis, papillitis and neuroretinitis carry the suffix *itis* to indicate an inflammatory process. While from the intracranial lesions there result most often a pure edema of the nerve head; in inflammation of the nerve there may be no disc changes whatsoever, or there may be edema even to the extent of the two or more diopters implied by the term choked disc, associated or not with appreciable inflammatory evidence. This article is concerned chiefly with the inflammatory type of optic nerve pathology called optic neuritis or papillitis, where there are disc changes recognizable, and retrobulbar or extraocular neuritis, where the inflammation of the nerve or sheath does not extend to the disc itself. There are many causes of such retrobulbar neuritis. Syphilis is a comparatively common and tuberculosis rather an infrequent factor in the etiology of optic neuritis. Various toxins or poisons circulating in the blood may give rise to optic nerve inflammation. This is exemplified by those cases resulting from a focus of infection or the excessive use of tobacco and alcohol. Actual invasion of the nerve by bacteria is an infrequent consequence of focal infection if we can judge by the tendency of the neuritis to spontaneous recovery. It is universally granted that the focus of infection may be in the tonsil, teeth, accessory sinuses or elsewhere. There may be no other symptom of retrobulbar neuritis than blurred vision produced by a central scotoma. On the other hand, the acute inflammatory character of the attack may be evidenced by pain

in the brow and about the eye, distress on movement of the eye, pain on pressure on the eyeball and inflammatory or edematous changes in the nerve head. While in syphilitic neuritis there may be some involvement of the periphery of the visual field, retrobulbar neuritis is usually characterized by a field normal except for enlargement of the blind spot with central scotoma for white or colors. The fields occasionally may be narrowed. The affection is usually onesided, but both nerves may be involved.

While the ophthalmologist has little difficulty in diagnosing the condition, the recognition of the etiologic factor in retrobulbar neuritis is beset by many difficulties and is of the utmost importance from the standpoint of therapeutics. Intracranial lesions, syphilis, tuberculosis, toxic alkaloids and other poisons must be ruled out of the picture. Disseminated sclerosis, of which retrobulbar neuritis may be an early and solitary symptom, is more difficult to dispose of.

Sinus infection, especially disease in the posterior ethmoids and sphenoid, has long been considered the most probable cause of retrobulbar neuritis. It has been held that the infection passed directly from the sinus to the adjacent nerve, causing inflammation of the sheath or of the nerve itself. Operations on these sinuses have been followed so frequently by immediate recovery that the etiologic relationship was thought to be proven until later developments showed that a large proportion of such cases recovered spontaneously. Also the findings in the sinuses at operation frequently failed to furnish convincing evidence of disease. Also if optic neuritis depended upon the infected sinuses, one would expect to find it more often associated with the extreme hyperplastic, polypoid, purulent disease so infrequently associated with it. In answer to this discrepancy it has been suggested that the chronic nature of the disease in the sinus builds up protective factors that prevent involvement of the nerve. Markbreiter,<sup>5</sup> in Onodi's Clinic, reported visual field changes in 70 of 100 cases of chronic sinusitis where there was no eye complaint or visual disturbance. Sometimes operation has been followed by cure, and again by disappointment when the pathologic findings were inconsequential and more or less identical. The mere blood letting has been assigned as the cause of the betterment.

Leon White believes that the neuritis is due to toxins carried in the blood from a focus of infection elsewhere in the body. While he admits the sinuses, especially the antrum, as possible sources of infection, he finds the tonsils and teeth furnish the toxin more often. He stresses the importance of a thorough neurologic examination and then proceeds to eliminate the most probable foci. He gives evidence to prove that there is greater danger to a nerve with an optic canal that is smaller than normal. He believes that recovery should take place in about 85 per cent of all cases, without opening the posterior ethmoids and sphenoid. Also in the 15 per cent of cases where he admits more radical measures are indicated, he merely attempts to aerate the sinuses. In reporting 60 cases,<sup>6</sup> the removal of the focus was followed by "normal vision in 50 per cent, marked improvement in 25 per cent." That leaves 25 per cent apparently not benefited to a satisfactory extent. Meller<sup>7</sup> believes the sinuses are the most frequent cause of the disease in the nerve and that the rhinologist should open these cavities on the recommendation of the ophthalmologist, independent of rhinologic evidence of disease. Meller's experience makes his opinion of great worth, but his dictates are opposed by Hajek,<sup>8</sup> who feels that the question is still open for solution. Van der Hoeve<sup>9</sup> says that the origin of retrobulbar neuritis is manifold and the relative percentages of various causes of the condition vary in different countries and cities.

After reviewing the subject of retrobulbar neuritis, one is struck by the difference of opinion that exists and realizes the futility of argument based upon personal opinion rather than upon pathologic facts. It must, of course, be granted that there are other foci responsible for neuritis, but the close association between the optic nerve and the posterior ethmoid and sphenoid sinuses puts the burden of proof upon those who look farther afield for the cause of the infection. It is too soon to rule out the sinuses because the operator saw nothing with his naked eye. It is also too soon to say that the apparently harmless inflammatory changes in the mucosa shown by the microscope are unimportant. Cytologic study of the antrum content has taught me that chronic inflammation can be present without any changes in the mucosa that can be recognized by the naked eye. I find repeatedly round cells and occasion-

ally polymorphonuclear leucocytes in the antrum where the X-ray is negative and the wash water, to the naked eye, absolutely clear. The centrifuge and the microscope may be necessary to collect and detect the evidence of chronic inflammation in the sinus which is in a semiquiescent state. I do not wish to give the impression of arguing in favor of the sinuses as a frequent cause of retrobulbar neuritis. I only believe that we should not disregard them as a possible cause until our knowledge is founded upon a more scientific basis.

Most surgeons are in favor of conservative treatment because of the frequency of spontaneous cure. But cases in considerable numbers are presented where a part or all of the vision is lost. The argument is also used that the opening of the sphenoid and posterior ethmoid have failed to cure cases of postorbital neuritis. How can we expect a cure to result from simple opening of a sphenoid or ethmoid sinus when the extension into the orbital roof and lesser wing of the sphenoid are the parts that actually lie in approximation with the nerve? These cells cannot be reached by simple aeration of a sinus or by any intranasal operation. Also it is not fanciful to conceive that failure to cure may be the result of the constriction of an inflamed nerve in a narrow optic foramen. Leon White has repeatedly called attention to the fact that the prognosis is worse where the canal is abnormally small. In a given case of postorbital neuritis there should be close cooperation between the ophthalmologist and the rhinologist. The neurologist and the internist or other specialist should be made available where advisable.

The presence or absence of sinus infection should be established. The perusal of literature would lead one to believe that the determination of such a diagnosis is very difficult; that it requires many observations and much searching in the patient's nose. As a matter of fact, the diagnosis of the presence or absence of sinus disease is easy and is not entirely dependent upon the examination of the nose, but is established by examination of discharge from the nose or nasopharynx of the patient. While in certain cases the discharge in a sinus is prevented from escaping, this we must recognize as a rare condition, accompanied by such evidence of accumulation and

pressure that diagnosis is not difficult. Practically every infected sinus gives rise to more or less secretion. This secretion makes its way into the nose or nasopharynx and can be detected.

I have believed for a long time and last year made the statement before this Society<sup>10</sup> that normally there are no lymphocytes or polymorphonuclear leucocytes in any considerable number in the mouth, pharynx, nasopharynx, nose or sinuses of an absolutely healthy individual. Such cells can be accounted for in the mouth and pharynx, or even nasopharynx, by pathology in the teeth, tonsils or adenoid, and in the nose by ozena, foreign body or other such condition. In practical, everyday work such cause for lymphocytes and polymorphonuclear leucocytes in numbers can be easily excluded. The fact is that accumulations of lymphocytes and polymorphonuclear leucocytes found on swabbing the nose or nasopharynx gently and examining a smear under the microscope are usually indicative of sinus infection. A simpler method of arriving at a diagnosis of sinus infection is to have the patient bring on a handkerchief the discharge from the nose or nasopharynx. Purulent or mucopurulent discharge can be recognized even when the saliva has dried away, by its color or glazed consistency. Discharge from the nose, containing lymphocytes or polymorphonuclears in numbers, either collected anteriorly or from the nasopharynx, is *prima facie* evidence of disease of a sinus in the absence of obvious nasal pathology to account for it. It is often difficult or impossible to get a history from the patient of discharge from the nose. There is a firmly rooted prejudice in favor of the fact that the nasal discharge is a "natural." While perfectly willing to admit that they have frequent or constant "colds," patients are prone to deny the presence of such a thing as "discharge" from the nose.

A case referred a few months ago is in point. The woman, a very intelligent person, denied all nasal discharge, even after persistent insistence on my part. I asked her nurse to go to her room and bring me her handkerchief, and on one of these was some definite, yellow mucopus that I found later came from the side of the nose corresponding to the eye affected with retrobulbar neuritis. With the ophthalmologist I concurred in advising an operation on the sinuses. This was refused, and



the very next day the eye began to improve. She is still conscious of a slight positive scotoma, but vision is practically normal.

Although the presence of discharge is diagnostic of sinus disease, it does not indicate which individual sinus is affected. The methods of determining this are outside the scope of this paper, but I put in the front rank for reliability the cytologic examination of the sinus content.<sup>11</sup> I believe that newer and better methods of diagnosing sinus disease will upset former statistics and show that sinus infection is present much more frequently in these cases. Even in the presence of homonomous sinusitis the question of operation demands the weighing of all the evidence of various foci of infection. The urgency of the case as outlined by the ophthalmologist must influence the judgment of the rhinologist. Where no evidence of sinusitis is present, more consideration can be given to the other possible foci of infection. I am firmly of the opinion that experience, as equivocal as it has been, warrants operation on the sinuses where, other therapy failing, the ophthalmologist determined that the nerve is being progressively damaged and there is discharge from the nose.

Having reviewed authoritative opinion as to operative indications, I hope I will not be understood to be advocating undue interference in presenting a very radical, technical plan. It is interesting to me as opening new operative avenues. I have removed the thin plate of bone between the optic nerve and the sphenoid sinus, under local anesthesia, in the living patient—a young girl of 14—with satisfactory result and no untoward effect. The complete opening of the optic canal as hereafter described, I have only worked out on the cadaver and present here merely as an anatomic study. I have felt encouraged to present this study by my experience following the reading of such a paper before this Society in 1926. I, at that time, outlined, a new transantral approach to the pterygopalatine fossa.<sup>12</sup> Shortly after my return home I was gratified by having my colleague, Dr. Harold Fletcher,<sup>13</sup> make use of that route and eradicate a large endothelioma from a patient, who is alive and well today.

Operation.<sup>14</sup>—Anesthesia, scopolamin and morphin sulphate, novocain 1 per cent injected, and cocain crystals applied intra-

nasally. The incision is the same as I have used in the ethmo-sphenoid-frontal operation under local anesthesia, but that technic I have modified to make a skin mucous membrane osteoplastic flap to prevent the closing of the frontal sinus opening by soft tissue retraction. The ethmoid mass is exposed through this flap and the ethmoid arteries are tied as heretofore.

The lamina papyracea is removed in the upper part after the ethmoid cells lying medially to it have been opened and studied. All supraorbital extensions of the ethmoid cells in the lateral mass and in the palate bone are opened and the orbital wall removed to allow their obliteration.

The sphenoid is opened and the wall between it and the depth of the orbit removed. This quickly leads to the location of the optic nerve, which shows plainly in the depth as the periosteum of the orbit, including muscular attachments, is separated from the bone. The thin bone between the sphenoid and the optic nerve is removed very carefully with Jansen-Middleton forceps. In a certain type of sphenoid with small sinus, this would be very difficult or perhaps impossible. However, in the great majority of cases it is feasible. If it is deemed advisable to definitely open the whole canal, it is necessary to lay the dura bare. This adds to the hazard of the operation, but in itself is not dangerous if we can be guided by such exposure in other operations. I cite, for example, mastoid work where the dura is commonly exposed. All of the other methods for reaching the optic canal which I noted included exposure of the dura in a much more radical fashion.

The dura is laid bare by removing the thin, bony plate in the upper, inner side of the orbit. (Fig. 1.) This opening is extended backward, and in this way the thin bridge of the lesser sphenoid wing comes into view. This is cut through with great care to prevent penetrating the dura on the posterior edge of the lesser wing of the sphenoid. This wing may be hollow and contain an extension of the sphenoid sinus and be the site of the disease that is doing most of the damage. When the upper and inner walls of the canal are removed there is no longer any possibility of the pinching of the nerve and all sources of sinus infection are removed from its course.

The optic chiasm lies exposed to view. Care must be taken to keep anterior to the nerve because of the carotid artery and hypophysis which lie immediately posterior to it. (Fig. 2.) There is no bleeding, due to the ligation of the ethmoid arteries. The osteoplastic flap is turned back into place and the wound fastened by metal skin clips. These have an advantage in this region because they do not extend through the skin where a suture might pick up infection and cause a stitch abscess. No dressing or bandage is applied.

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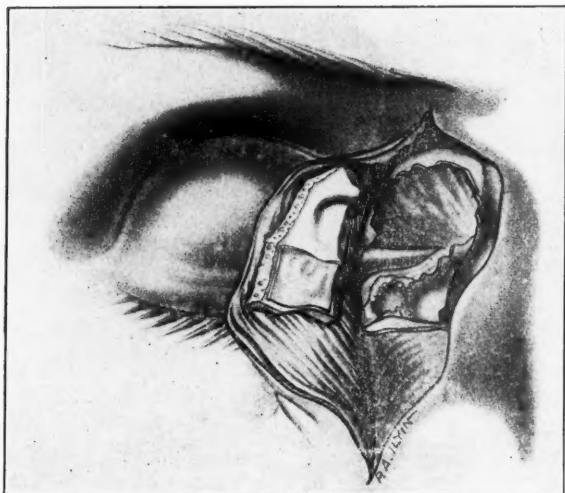


Fig. 1. Osteoplastic flap covered by skin and mucosa. In the depth is seen the optic nerve. Above the dura covering the frontal lobe has been exposed by removal of orbital roof. This bone has been removed backward to include the lesser wing of the sphenoid. The dark sulcus seen above the nerve was occupied by the lesser wing. The depth of the sphenoid sinus shows below the nerve which lies freely exposed from the optic canal well on to the optic chiasm.

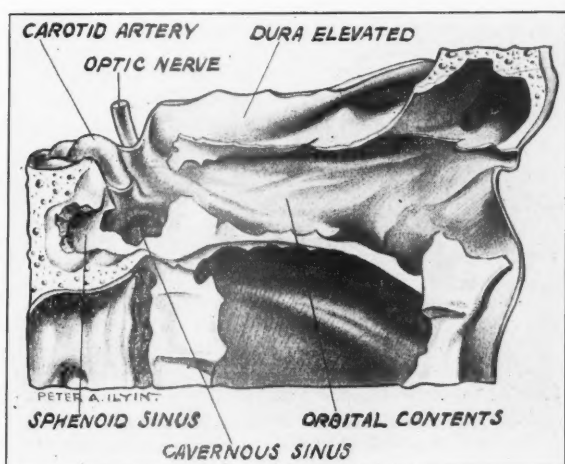


Fig. 2. Orbital contents exposed from the medial side. The dura is elevated and the sulcus above the optic nerve, formerly occupied by the lesser wing of the sphenoid, is seen. This dissection shows the proximity of the carotid artery.

## LXVI.

### SOME NOTES ON THE DIAGNOSIS AND SYMPTOMATOLOGY OF BRAIN ABSCESS OF OTITIC ORIGIN.\*

By JAMES A. BABBITT, M. D.,

PHILADELPHIA.

As indicated by the title, this paper will review some phases presented in the diagnosis and symptomatology of this trying type of case. While suggesting certain phases for discussion appropriate for group consideration, and by the title making this possible, its real purpose is to record their incidence in one particular case with rather remarkable recovery, and briefly review some points which will at least bear repetition if not add further light. More detailed hospital notes, charts and X-ray studies will be appended to make the report more complete.

Briefly the story of the case selected for the text is as follows: This young man, John S., 35 years of age, was sent into the Lankenau Hospital after about three weeks of irregular illness—with moderate temperature, not over two or three degrees, and his physician's diagnosis of left nasal sinusitis, probably antral. His previous eight days had been in bed, somewhat delirious and irrational. His left ear had been having slight discharge, following an attack of scarlet fever, to which he gave absolutely no importance nor reference of pain for twenty-seven years.

The impression given to us at the hospital was that of an interesting rather than a very ill patient. His physician, a clever practitioner, must have sensed its seriousness, however, for he sent him in by ambulance, and, as a matter of fact, he staggered when attempting to walk and was unable to read at that time.

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Although he arrived late Saturday afternoon, an X-ray study of sinuses was obtained, and interpretation by wet reading eliminated the accessory sinuses. There was, however, a nasal deflection which nearly blocked his affected side.

Within the first thirty-six hours, five important studies were made and opinions given.

The internist, after studying his pain reference, left frontal or in the neighborhood of the external canthus of the left eye, detecting a faint suggestion of irregularity of pupils and sluggishness of left facial muscles, general dullness, yet absence of meningeal indication, gave a prompt and brilliant diagnosis of either brain tumor or brain abscess.

The ophthalmologist pronounced the fundus changes as practically negative.

The neurologist reported that the patient's lack of cooperation and recent use of mydriatic rendered eye symptomatology negative—character of cells in spinal fluid ruled out syphilis; sugar in fluid argued against infective meningitis. Ear condition rendered temporosphenoidal or cerebellar abscess worthy of consideration.

The X-ray department reported all nasal sinuses clear; cloudiness of left mastoid; haziness of right.

The laboratory reported a leucocyte count of 16,300. Hemoglobin 75, neutrophiles 69, lymphocytes 25. Urine practically negative. Spinal fluid examination, cloudy, globulin slightly positive. Sugar (Fehling reduced). Cell count, 1500 per cu. mm. Neutrophiles, 66 per cent; lymphocytes, 30 per cent; eosinophiles, 4 per cent.

The otolaryngologist felt an inclination to surgically investigate the left mastoid area.

On Monday, August 15, 1927, forty-eight hours after admission, patient was operated upon under nitrous oxid, oxygen and ether. A completely eburnized mastoid area was tediously excavated; practically no antrum cavity was found. The sinus was exposed and walls appeared normal. Through the mastoid tegmen an exploration reached a large temporal lobe abscess, about three inches in diameter to probe, and yielding about 60 cc. of foul smelling pus, culturing to gram positive cocci, diplococci and bacilli, according to laboratory report.

The patient had been completely comatose some hours before the operation, but stood the tedious procedure reasonably well. Four hours later had a five minute chill; pulse rapid and weak; temperature rose to about 102; axillary pulse to 132, and respirations 36. By the following morning had materially brightened, pulse, temperature and respiration down, and part of the time rational. The following day stupor had returned, aphasic symptoms, rigidity of neck and right side of body occurred, and, assuming faulty drainage, the incision was enlarged by an associate and double tube drainage installed.

For five days conditions were extremely unfavorable—stupor, aphasia, increasing hemiplegic symptoms, incontinence, frequent periods of Cheyne-Stokes respiration but no rise of temperature or pulse. On the eighth day after the primary operation his temperature suddenly shot up four degrees and all symptoms became worse. Considerable laboratory study was made. Spinal cell count was down to 35 per cu. mm., smear reported gram positive cocci and diplococci. Blood culture was now reported negative after three days' incubation, Wassermann negative. Spinal fluid pressure registered 30 mm. On applying pressure over right jugular unaffected side, in taking Queckenstedt test there was a rise of 10 mm.; on the side operated upon no change was noted in reading. This indicated, of course, sigmoid sinus disturbance from pressure or infection. So the patient was again sent to operating room, lateral sinus opened and explored. There was no definite thrombus, though there must have been a thrombophlebitis. The external jugular was tied off and the sinus cavity packed. Conditions immediately improved.

This ends the story. After a long and somewhat checkered convalescence the patient left the hospital practically well, and today—after three major operative procedures and the intervening well nigh moribund symptomatology—is working as foreman again in his old position.

This brief history, and similar conditions in other brain abscess case records, suggest certain phases of importance for discussion.

First. The Obscure Etiology and Difficulties in Clinical Diagnosis.—The time honored expression that "when a brain



abscess is recognized it is too late to operate" renders the judgment as to justifiable interference one of paramount importance. Hundreds of patients live patiently with a chronic intermittent discharging otitis media, the very chronicity of which has bred a resentment to interference. Now and then one insidiously develops an intracranial lesion, but the diagnostic indication of this may be hopelessly obscure, and might almost be described as an "aura of impending trouble." Doubtless many of us sorrow in the recollection of failure to interpret this incubation stage.

The important thing in this particular case was that the left ear, though not troubling the patient, had been in trouble for twenty-seven years. Our first examination shared his indifference to it, as it did not even suggest chronic mastoiditis.

This well advanced abscess, with naturally a surrounding cerebritis, presented no eyeground, no disturbed reflex, no meningeal evidence, which could not be referred to the weakness from a three weeks' illness of the semi-influenza type. The X-ray examination eliminated active sinusitis, and revealed a sclerotic mastoid. How sclerotic this was, the operative tedium well revealed.

Second. Neurologic Examination.—The most significant fact perhaps in the neurologic examination by both specialists was the elimination of other factors which would indicate more than a localized condition. The aphasia and rigidity of the right side and semi-hemiplegic condition which was indicated more clearly following the first operation, are both significant in localizing the trouble and also in indicating that it was thoroughly walled off from the surrounding areas.

It is a great satisfaction after eight months to find that the dire results predicted from the invasion of so large a brain area have not pertained in the patient. He is not distinctly normal but he has no definite aphasia or apraxia, or any motor weakness at the present time. He does not suffer from incoordination or any ocular difficulty.

Within the last month the writer has had occasion to observe a similarly located temporal abscess, seen in consultation, in which the aphasia and hemiplegia, somewhat more marked, were almost identical. This did not follow an otitis media but followed a left orbital infection and appeared shortly after

some major sinus operative work on that side for its relief. Curiously the Queckenstedt test in this case also showed failure in transmission through the left sigmoid. The writer was able to see the autopsy upon this patient—a left temporal abscess almost equal in size, apparently fairly well walled off and no thrombotic evidence in the sigmoid or the cavernous sinus. One suspects that here, too, there may have been a thrombophlebitis.

Third. Operative Procedures (in first patient).—In the light of subsequent developments, though the opening through the tegmen was enlarged to at least 2 cm., and this seemed ample drainage, it was evidently not sufficient. The second enlargement was doubtfully sufficient. The sinus involvement may have been due to direct extension and may have been incidental to the pressure of the tubes in the large incision. Several conference opinions were expressed as to procedure during this stuporous stage. The writer is inclined to believe that the decision, largely influenced by the opinion of Dr. Shoemaker, to avoid additional trephining and to persist in simple saline drainage, probably saved the patient's life, though it is doubtful whether he would have lived without walling off the jugular vein.

Fourth. Remedial Measures.—The question of glucose administration will perhaps raise discussion. In the presence of a cerebritis, dehydration should prove of value. In an associated meningitis, which probably was not seriously present, the opposite would hold. The resident, Dr. Engel, administered intravenously first, immediately after the operation, 50 grams with 1,000 cc. saline; constantly administered it by bowels in connection with whisky and saline; and on ten different occasions 20 cc. of 50 per cent glucose intravenously. One cannot but feel that this was an important factor in his recovery.

Fifth. General Observations on the Case.—It is a matter of interest to note from what severe operative procedures a patient can be expected to recover. This patient had three major operations of considerable duration each, which must have been a severe shock. He had a week of complete comatose type of prostration, which included Cheyne-Stokes respiration and in which there seemed little semblance of favorable reaction. Sin-

gularly enough, during this time there was no great rise or fall of temperature, pulse and respiration.

It is at least a comforting reflection to find that such an aphasia, such a marked involvement of the right side, such continued coma and lethargy, did not interdict a complete recovery, and it is largely to insert notes of encouragement in regard to this that this brief paper has been undertaken.

#### CONCLUSION.

1. Remote facts in etiology and slender diagnostic indications may justify major brain abscess surgery.

2. The lack of correspondence in temperature and pulse lines, general therapeutic picture and lack of conformity to conventional laboratory values are often characteristic of brain abscess.

3. Positive localization and negative elimination have the same relative value in ophthalmologic and X-ray findings in these cases.

4. The spinal fluid pressure test of Queckenstedt-Tobey types should be a more routine procedure and may often save life.

5. The study of aphasic and hemiplegic symptoms may determine the value of the dehydration glucose treatment.

6. One is surprised to note the degree of continued prostration and coma as well as extent of operative interference from which a brain abscess case may survive.

#### SOME NOTES FROM THE CASE RECORD.

##### GENERAL HISTORY.

J. F. S., married, 35 years of age, an American; occupation, patternmaker. Admitted to the Lankenau Hospital August 13, 1927. Referred as a patient with probable ethmoid sinusitis on the left side. July 28th, sixteen days ago, he commenced to have a temporo-frontal headache. This was considered influenzal—was attended with some fever. Patient remained in bed four or five days, then returned to his work as a patternmaker. After two days, returned to bed because of fever and headache. Remained in bed from August 5th to admission, August 13th. During this time was irregularly delirious and irrational. Temperature was variable, never above 101 F. Has been staggering somewhat when attempting to walk. There has been no history of vomiting or stiffness of the neck. No congestion of the chest, hemoptysis, cardiac pain, nocturia, hematuria, nasal discharge or tonsillitis. Patient had scarlet fever

in 1900, leaving an intermittent discharge in the left ear. The hearing was poor, though he refers no particular trouble to it. Appetite has been variable, bowels constipated during recent illness. Also had typhoid fever when fifteen years of age, and severe attack of influenza in 1918. The family history was entirely negative as relates to this condition.

#### EXAMINATION ON ADMISSION.

Patient lies quietly in bed with no great discomfort, somewhat mentally befogged, and history was given by the wife.

General: Heart, lungs and abdomen negative. Blood pressure 92—46.

Ears: There is chronic infection of the middle ear, with slight amount of mucopurulent discharge. The right ear is negative.

Eyes: Pupils equal; react sluggishly to light and in accommodation. No ptosis, palsies, exophthalmus, or lid-lag. No nystagmus.

Nose: Septum deviated to left, with poor passage on left side. Left antrum somewhat cloudy.

Mouth: Tongue protrudes in midline. Coated white but moist. Teeth in poor condition. Tonsils quiet.

Neurologic: No rigidity of neck. Knee jerks exaggerated. Babinski and Kernig negative.

Blood examination: R. B. C. 4,410,000, W. B. C. 16,300. Hb., 75 per cent. Neutro 69; lympho 25; L. mono 6; trans 0; eosin 0.

Urine examination: Reaction acid; specific gravity 1021; no albumen or sugar; diacetic positive; some crystals, epithelium and mucous.

#### SPECIAL EXAMINATIONS.

August 13, 1927. X-ray examination: All sinuses appear clear. Cloudiness of left mastoid, haziness of right.

August 14, 1927. Medical examination—Dr. Harvey Shoemaker: From location of pain, very slight inequality of pupils, a little motor sluggishness of the left side of the face, general stupor and patient's appearance, provisional diagnosis of probable brain tumor or brain abscess was given.

August 14, 1927. Eye examination—Dr. W. T. Shoemaker: Patient stuporous and nonresponsive. Left palpebral fissure a trifle narrower than right. (May not be a new condition.) Eyes are straight in primary position. Ocular movement cannot be detected. Ophthalmoscope shows both eye discs well defined, vessels good, no changes. Examination practically negative.

August 15, 1927. Neurologic examination—Dr. Chas. S. Potts: Pupils unequal. Left smaller than right. Right pupil contracts to light, then rebounds. Left pupil did not contract to light. Mydriatic had been used recently. Patient is stuporous and will not obey commands and will not cooperate. Otherwise negative. Character of cells rules out syphilis. In view of ear condition and symptoms, possibility of an abscess being present should be considered. Noncooperation of patient makes diagnosis of abscess in temporosphenoid region of cerebellum rather difficult. Sugar in fluid argues against infective meningitis.

August 19, 1927. Neurologic examinations—Dr. Temple Fay: "There was a weakness of the right arm, and slight of the face, with increase of reflexes but no Babinski or clonus. The rigidity of the neck and Kernig were marked, but there appeared to be no evidence of cortical irritation, such as purulent meningitis, as there was no convulsive movement or differentiation of the eyes to the left.

"Since instituting drainage and irrigation of the cavity which you have so successfully found and drained, I have seen him and noted his improvement. There will undoubtedly be a residual aphasia and lethargy for some days which may never clear, but at the present time it looks hopeful. The rigidity of the right arm has decreased, and the patient responds better to stimulation than formerly.

"I feel that the intravenous glucose has assisted in partially clearing the cortical condition and hope that he may survive.

September 2, 1927. "At the present time (September 2nd) the patient is bright, understands some things, and is able to express himself slightly. The rigidity has left the right arm. Reflexes have returned on the right. Babinski has disappeared. An attempt to determine hemianopsia has not been possible. There is slight weakness of the left face, but no other neurologic manifestations. Stiffness of the neck has greatly decreased. The symptoms are those of a deep left temporal lobe lesion, and, when cooperation can be secured, visual field examination may give the extent into the region of the optic radiations."

#### SPINAL FLUID EXAMINATIONS.

August 15, 1927. Cloudy, globulin slightly positive. Sugar present (Fehling reduced.) Cells 1500 per cu. mm.

August 19, 1927. Cell count 35—all lymph. Smear shows gram pos. cocci and diplococci.

August 23, 1927. Cloudy. Cell count, W. B. C. 6940; polynutr. 44; lymph. 27. L. mono. 25; trans. 4; eosin 0. Occasional gram pos. cocci (diplococci of pneumococcic type). Globulin, negative for excess. Sugar, slight reduction, Fehling 1/1.

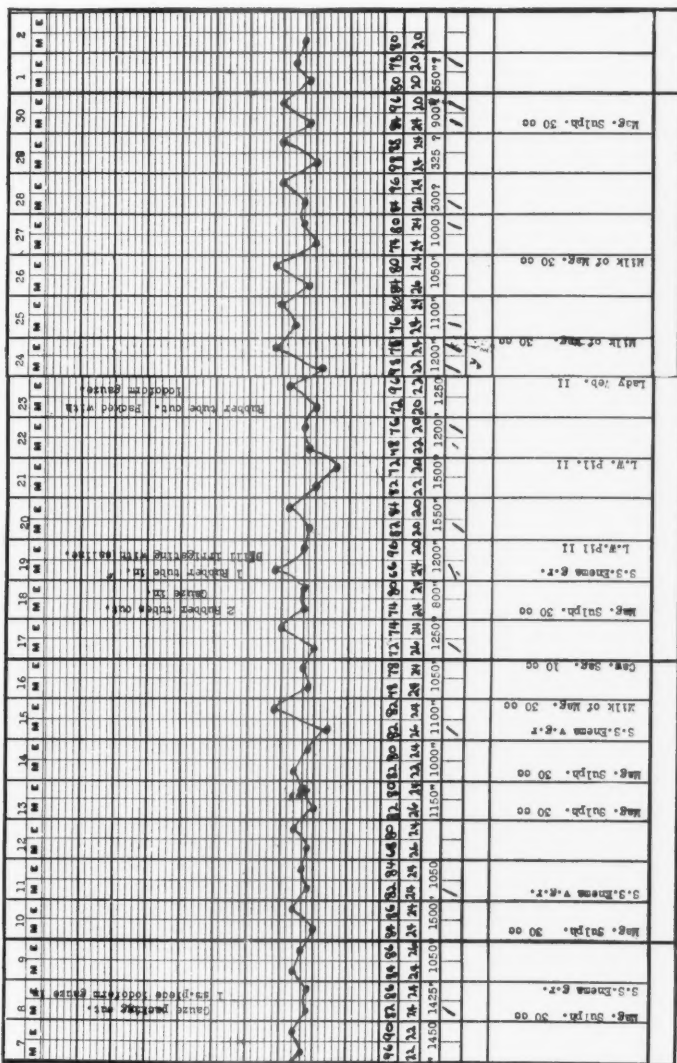
Queckenstedt test: Pressure right jugular, mercury rise 10 mm. On release of pressure, mercury dropped rapidly. Pressure of left jugular, no change in column.

#### NOTE ON BLOOD AND URINE EXAMINATIONS.

There was no great variation in the blood examinations, R. B. C. ranging around 4,000,000; highest W. B. C. count, 16,300; highest neutrophile, 81; hemoglobin, from 75 to 85; Wassermann reported negative and blood culture sterile.

Urine examinations gave specific gravity ranging from 1007 to 1033; no sugar; faint traces of albumen; practically no casts; occasionally white blood cells; acetic and diacetic acid only present in early stage.

| DATE | TIME | TEMP | PULSE | BLOOD PRESSURE | RESPIRATIONS | WEIGHT | HEIGHT | HAIR | SKIN | TEETH | NOSE | THROAT | STOMACH | INTESTINES | BLADDER | RECTUM | GENITALS | NEUROLOGICAL | PSYCHIC | GENERAL | REMARKS |
|------|------|------|-------|----------------|--------------|--------|--------|------|------|-------|------|--------|---------|------------|---------|--------|----------|--------------|---------|---------|---------|
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   | 14   | 13   | 12    | 11             | 10           | 9      | 8      | 7    | 6    | 5     | 4    | 3      | 2       | 1          | 0       | 0      | 0        | 0            | 0       | 0       | 0       |
| 15   |      |      |       |                |              |        |        |      |      |       |      |        |         |            |         |        |          |              |         |         |         |



LXVII.

PERSISTENT HICCOUGH: CONTROL BY COCAINIZATION OF THE NASAL (SPHENOPALATINE, MECKEL'S) GANGLIA.

BY JAMES B. COSTEN, M. D.,\*

ST. LOUIS.

Hiccough is generally described as an affection of the phrenic nerve, giving rise to a clonic spasm of the diaphragm. The sudden inspiration produces a closure of the glottis, with a sharp ("hic") sound, as a relatively large volume of air is drawn through the narrow laryngeal aperture. Osler<sup>1</sup> considers "the afferent impressions to the respiratory center as either peripheral or central, the efferent distributed through the phrenic branches to the diaphragm, causing the intermittent spasm, and through the laryngeal branches of the vagus to the glottis, causing sudden closure as the air is rapidly inspired." A common experience demonstrating this reflex is the production of a mild hiccough by "clearing the throat" forcibly.

It seems reasonable to group these phenomena as of their peripheral or central origin.

The attacks of peripheral origin would include intimate irritation of the diaphragm from excessively full stomach, ingestion of hot, cold or gaseous fluids; and from more serious inflammatory causes, the cases associated with peritoneal irritation, as peritonitis, gastritis, hernia, appendicitis, and severe forms of enterocolitis.

Those of central origin include cases in hysteria or emotional crises; organic brain disease, as apoplexy, tubercular meningitis, or brain tumor, show the reaction as a terminal symptom; blood borne poisons irritate the respiratory center, as in diabetes, chronic nephritis, during influenza or after the attacks; Purves-Stewart<sup>2</sup> describes hiccough as sometimes due

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to the toxin of epidemic encephalitis, and in this connection observes the spasms to involve not only the diaphragm but also the muscles of the abdominal wall, unilaterally or bilaterally.

Rosenow<sup>3</sup> accounts for epidemic hiccough as a close corollary to epidemic encephalitis, and reproduces in animals the symptoms of both diseases, by injection of organisms from nasopharyngeal washings of patients with epidemic encephalitis, and epidemic hiccough. He reports prompt relief of spasms of the diaphragm and hiccough in a number of patients and animals following the use of encephalitis immune serum.

Treatment of hiccough includes infinite varieties of reflex irritations, as causing a sneeze, holding the breath, swallowing ice, salt or sour substances, lavage of the stomach, chilling, faradization or application of irritants to the hypogastrium. Apomorphin is often used, and Osler states that most effective of all is the use of large and repeated doses of morphin. Direct effort on the phrenic nerve includes pressure on the carotid sheath, galvanization over the carotid sheath (Friedenwald and Levy<sup>4</sup>), and section of the phrenic nerve by various investigators. Rhythmic traction of the tongue is reported effective in cases of Laborde and Noir.<sup>5</sup> Sluder<sup>6</sup> refers to four cases of Rehfeldt and one of Hansel, in which intractable hiccough was controlled by cocainization of the nasal ganglia. Sluder having included the control of hiccough through the nasal ganglion as a rhinologic problem, has directed considerable interest to this possibility. Most attacks of hiccough subside without incident, usually after a simple remedy. It is only the serious, persisting case which comes under elaborate observation and treatment.

The following case, a patient of Dr. C. H. Eyermann, showed uncertain control by all remedies used, and definite, sudden halt of the contractions when the nasal ganglia were cocainized.

August 14, 1927. H. P. K., 55, entered Barnes Hospital suffering from strong hiccough contractions of five days' duration, rate from 18 to 22 per minute.

Family history: M. D., 83; senility. F. D., 46, of accident. One brother, dead of acute nephritis.

Past history: For a number of years he has had precordial pain with attacks of indigestion. During this time he has been

nervous and irritable, and during excitement has had attacks of nausea. He has occasional pain in the epigastrium and fainting spells. He has had a right inguinal hernia for 34 years. One attack of hiccough, eight years ago, was relieved by chloroform anesthesia.

Present illness: Five days ago, after a full meal, which included a bottle of homemade beer, he began to hiccough violently. At intervals since, he has had severe vomiting spells. No food has been taken these five days, because he cannot swallow. He has had no relief from gastric lavage, morphin, enemas or other efforts.

Physical examination revealed considerable shock from starvation, varicose veins over legs, and a large right inguinal hernia.

August 16, 1927 (Dr. Duden).—Three days in hospital, includes use of morphin and atropin, paraldehyde, chloretone, Hoffman's anodyne, nothing by mouth, and the use intramuscularly of 2 cc. of 50 per cent magnesium sulphate with morphin-atropin.

August 17, 1927 (Dr. Duden).—Following the hypodermic of magnesium sulphate, morphin-atropin, the patient slept continuously throughout the night, but hiccoughed about 20 contractions per minute during sleep. Patient is very exhausted.

August 17, 1927 (Dr. Costen).—Both nasal ganglia cocaineized, with saturated solution, removed after one minute because patient showed signs of intoxication, clammy perspiration, rapid pulse and choking. The violence of the hiccough subsided promptly.

August 17, 1927, P. M. During the day the hiccough ceased entirely for one hour, at three different periods, then resumed. Dr. Duden dilated anal sphincter with three fingers, and change to slower rate was only effect noted. Nasal ganglia were again cocaineized, ten minutes, risking intoxication. The instant that the second applicator was in place the hiccough stopped and did not recur for five hours. Promptly after it stopped, morphin, grs.  $\frac{1}{4}$ , hyoscin grs.  $\frac{1}{130}$ , were given as hypodermic, hoping to continue the relief obtained.

August 18, 1927 (Dr. Costen).—Both ganglia cocaineized twenty minutes. Contractions stopped suddenly, as before, when application was made. Twelve hours' relief.

August 18, 1927. Ganglia cocainized twenty minutes; prompt halt of symptom; no further recurrence except an occasional mild contraction for a few minutes.

August 24, 1927 (Dr. Duden).—No further signs of recurrence for five days, and rapid gain in strength since feeding became possible.

August 26, 1927. Discharge Note (Dr. Eyermann).—No gross pathologic findings from physical examination. The first lasting relief was obtained by cocainization of both nasal ganglia. Recocainization produced immediate relief with each trial and the interval of recurrence increased. The use of hyoscin-morphin narcosis seemed to produce further relief. Eating cracked ice stopped the hiccough a few minutes at times, and there were periods of cessation without any assignable cause.

#### SUMMARY.

The onset of this attack is definitely associated with distention of the stomach, with fluid of alcoholic nature, a most frequent original cause. This simple inciting factor, of peripheral origin, was removed undoubtedly by gastric lavage and enemas, the first treatment used. What continued the mechanism after such a factor was removed? All that apparently remains is the cycle of events in this hiccough.

The two movements in hiccough are the inspiratory excursion of the diaphragm and the closure of the glottis. This violent closure of the glottis may furnish enough sensory shock to precipitate the next contraction of the diaphragm. The effort in all kinds of treatment is to break up this reflex in the sympathetic system.

In this case and the others referred to, we have been able to interrupt the cycle by blocking the nasal ganglia, and it appeals to me as a corollary to the relief of laryngeal pain (Sluder<sup>7</sup>) by the same means; that sensation from the larynx may traverse this same route to set off the next spasm of the diaphragm, as accessory stimulus to the next reflex action.

If the original irritation of the diaphragm is peripheral, and removable, it seems that blocking of the ganglia would succeed in more cases than if the source of the spasm is central, where direct irritation of the phrenic nerve continues. Fur-

ther observation will show if there is any difference in results in the two classes of cases.

The cessation of hiccough did not occur until the second of the two ganglia was treated.

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## LXVIII.

### CEREBROSPINAL RHINORRHEA FOLLOWING INTRANASAL SURGERY.\*

BY EDWARD H. CAMPBELL, M. D.,

PHILADELPHIA.

The escape of cerebrospinal fluid following operations within the nose is apparently a very rare occurrence, as but few cases have been reported in literature. Spontaneous escape of the fluid as well as cerebrospinal rhinorrhea following injuries to the head, however, have been the subject of many articles during the past thirty years.

Sir St. Clair Thomson,<sup>1</sup> in 1899, reported a case of his own along with twenty others collected from the literature. He selected these cases as representing spontaneous escape of cerebrospinal fluid from the nose and excluded those cases which followed injuries or were probably due to intracranial tumors.

These twenty-one cases included nine in which the discharge was undoubtedly cerebrospinal fluid, and twelve in which the discharge was most probably cerebrospinal fluid. In addition Thomson collected eight other cases that were suggestive of an intracranial origin of the nasal discharge. Seventeen of his twenty-one cases showed cerebral symptoms and he considered that they might possibly have been due to internal hydrocephalus, having as its basis a serous meningitis. Post-mortem examination in four of the cases gave no evidence to disprove his hypothesis.

In a recent search of the literature, Locke<sup>2</sup> found records of fourteen fatal cases of spontaneous cerebrospinal rhinorrhea in which necropsy had been performed. Twelve showed internal hydrocephalus; the records of the other two were incomplete. In eight of these twelve the hydrocephalus was

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\*Read before the Section on Otology and Laryngology of the College of Physicians of Philadelphia, April 18, 1928. From the Department of Otolaryngology of the University of Pennsylvania.

due to cerebral tumor causing obstruction within the ventricle system. Two were cases of congenital hydrocephalus in which rhinorrhea developed in early adult life after the fontanels and sutures had closed, and two were cases of hydrocephalus of the adult type.

Eleven of these twelve reports showed that there were openings between the floor of the anterior cranial fossa and the nasal cavity. In the twelfth case only a thinning, and no opening, was found.

In 1926 Johnston<sup>3</sup> made a detailed report of twenty-one cases of all types of cerebrospinal rhinorrhea. These included one of his own and twenty collected from the literature published since 1900.

Since the publication of Johnston's article there have appeared reports of nine additional cases. Among these there have been but three that have been attributed directly to operations upon the ethmoid sinuses.

Probably the first case of the kind forming the basis of this report was one recorded by Williams<sup>4</sup> in 1913, a patient who had a cerebrospinal rhinorrhea complicated by a pansinusitis. The patient was a man, aged 42 years, who had noticed for about two years a swelling in the upper internal angle of the right orbit. He had been subject to severe headaches for years. Examination of his nose showed both nasal passages to be completely blocked by polypi but no purulent discharge. He had, however, noticed on several occasions a profuse watery discharge from the nose, accompanied by a subsidence of the swelling in the orbit. He was thought to have an occlusion of the ethmoid cells by the polypi with the development of an ethmoid mucocele. The polypi were removed and a bilateral intranasal antral operation performed. On further procedure a fistulous opening in the floor of the right frontal sinus was found. A Killian operation was next done on the right side, the nasofrontal passage enlarged and the left fronto-ethmoid cells of the frontal septum were removed. The dura was found to be exposed by a dehiscence of bone over an area of about three-quarters by one-half inch. The patient died of meningitis apparently due to infection through a small communication through the dura, exposed at the time of the

operation. The author concluded that the intermittent watery discharge from the nose was due to the escape of cerebrospinal fluid through the opening in the dura. This opening would become periodically obstructed by the polypi, with resulting accumulation of the fluid in the upper inner angle of the orbit. While this case was probably one of cerebrospinal rhinorrhea preceding rather than following intranasal surgery, it is, however, included among this collection of cases because the pathologic condition found was much similar to that found in some of the cases later reported.

Harvey Cushing,<sup>5</sup> in an article published in 1921, described a patient who had an acoustic tumor and who had had various intranasal operations leading to an ethmoidal herniation of the dura, followed later by cerebrospinal rhinorrhea and death. This patient, a man 41 years of age, had a bilateral exenteration of the posterior ethmoidal and sphenoid sinuses after a diagnosis of either brain tumor or hyperplastic ethmoiditis and sphenoiditis had been made. On the left side was found what appeared to be a dehiscence over the posterior ethmoid and the upper outer portion of the sphenoid wall, brain pulsation being clearly seen. Following the operation there was a persistent mucopurulent discharge from the left side of the nose with an increasing tumor mass apparently springing from the sphenoid and the posterior ethmoid region. This mass gradually increased in size and pulsated with the heart beat.

About a year and a half previous to this operation he had had his frontal sinuses drained (method not mentioned) and part of a turbinate and septum removed, and also a tonsillectomy. These operations were done because of persistent occipital headaches and a sense of pressure above the eyes, and were followed by an improvement for a time.

When the patient came into Dr. Cushing's hands he was studied thoroughly, both generally and neurologically.

Examination of the nose showed a slight watery nasal discharge which apparently had been present since his sphenoid-ethmoid operation. In addition was seen a pulsating mass far back in the left naris.

At operation a typical acoustic tumor was found in the right recess, capped by an unusually multilocular arachnoid cyst. A

partial intracapsular enucleation was done, followed by recovery with complete relief of the subjective discomfort.

Six months after the removal of the intracranial tumor, during a paroxysm of coughing, there was a sudden gush of clear watery fluid from the left nostril, followed thereafter by a persistent dripping. Several days later meningitis developed and the patient died. The autopsy disclosed a defect in the roof of the left ethmoidal region through which there was a dural protrusion. The protrusion contained a frontal lobe herniation and the leak communicated directly with the ventricle.

Cushing concluded that owing to the long standing hydrocephalus due to the subtentorial tumor the cranial bones had undergone some pressure absorption and that a pull on the wall of one of the ethmoid cells dislodged a considerable piece of the plate through which a herniation took place.

In 1923 Loftus<sup>6</sup> recorded the case of a woman forty years of age who complained of a persistent dripping of clear fluid from the left nostril. She had been treated for sinusitis and the dripping apparently started after a laughing attack, followed by severe sneezing. The discharge of fluid was accompanied by severe frontal headache extending to the vertex, the headache diminishing when the flow increased. Nasal examination showed marked congestion of the mucosa of the left nostril, slight hypertrophy of the inferior and middle turbinates and the absence of pus in the nose. The fluid came from the posterior part of the roof of the nose. The discharge of fluid subsided after six weeks in a hospital but two weeks after its cessation the patient suddenly became ill with high fever, numbness of the body and severe headache. She died three weeks later from acute encephalitis.

In this patient the rhinorrhea did not actually follow intranasal operation, but it did follow or accompany treatment for a sinus infection. Just what and where this sinus condition was, and what type of treatment had been given, was apparently not known by the author, but there may have been some relationship between the treatment and the occurrence of the discharge of spinal fluid. On the other hand, it may have been one of the type of spontaneous cases due to an intracranial lesion.



A case somewhat similar to this one was recorded two years later by Stein.<sup>7</sup> His patient was a woman, aged 45, who had developed a profuse watery discharge from the left nostril one year after both middle turbinate bones, which were hypertrophied and polypoid, had been removed with a snare. The flow of fluid was continuous and accompanied by a slight headache. An alcohol injection of the left sphenopalatine ganglion was given without result. Benzyl benzoate solution, twenty drops three times a day, was administered. After three weeks the discharge entirely stopped and had not reappeared in the following five months.

In 1926, Hunt<sup>8</sup> of London, Ontario, reported the development of cerebrospinal rhinorrhea following an ethmoidectomy. The patient, a man of thirty, had a history of nasal catarrh and a peculiar psychic disturbance, which caused him to be very irritable and quarrelsome. An X-ray examination showed cloudiness of the left posterior ethmoid cells and the sphenoid sinus. Under local anesthesia, the middle turbinate was removed and the ethmoid labyrinth, which was found to be soft and necrotic, was opened by means of gentle curetting.

The patient suddenly became faint and complained of severe frontal pain. High up in the nasal cavity a continuous trickle of clear cerebrospinal fluid was seen and also an area of the dura about a quarter of an inch in diameter. Hunt thought that the patient was doomed to a meningitis unless something was immediately done to prevent the spread of infection from the nose. He, therefore, rapidly opened the frontal sinus externally, and removed the posterior wall and adjacent bone over an area of about one inch in diameter. The dura was then elevated over the roof of the ethmoid until the dehiscence in the bone was detected. This perforation was then carefully walled off by means of a strip of iodoform gauze, the ends of which were brought out at the inner angle of the wound.

Following the operation the patient had considerable headache and some fever for a few days but this gradually subsided. On the seventh day the skin flap on the forehead was reopened and the gauze strip removed. Thereafter the patient's recovery was uneventful.

In 1927, Alden<sup>9</sup> reported another case of cerebrospinal fistula following ethmoidectomy. His patient was a woman of

forty, weighing 240 pounds, who complained of pain in the right side of the head following an acute cold. Examination revealed a marked superior deviation of the septum to the right compressing the right middle turbinate against the lateral wall of the nose. Pus could be seen in the right middle meatus. She was treated conservatively for acute sinusitis and had recovered at the end of a week. The headache, however, persisted, always located around and behind the right eye and in the right parietal region.

X-ray examination showed a cloudiness in the right ethmoid region. Under local anesthesia, a right middle turbinectomy was performed. When the anterior half of the turbinate and ethmoid wall were removed, a cavity was disclosed which apparently had been a closed empyema of the ethmoid bulla and contiguous cells. In the extreme medial superior part of this cavity a downward bulge was seen which proved to be dura. The edges of the incision were trimmed off and no packing was inserted.

The next morning it was observed that she had a discharge of cerebrospinal fluid from the right nostril. She was given saline cathartics daily and  $7\frac{1}{2}$  grains of urotropin three times a day along with a restriction of liquids in her diet. At the end of a week the dripping of fluid had apparently stopped and the patient's recovery was uneventful.

A few months ago, Collins<sup>10</sup> reported a case of cerebrospinal rhinorrhea following the rasping of a frontal sinus. After two applications of a rasp to the left nasofrontal duct there was a profuse rhinorrhea which was in all probability cerebrospinal fluid. Complete recovery occurred after eighteen days of rest in bed and palliative measures. X-ray examinations of the skull showed wide vessel margins suggestive of increased intracranial pressure. The author's opinion was that at the time the rasp entered the frontal sinus there was a herniation of the brain through an opening in the posterior wall of the frontal sinus.

The case which I wish to report is one of cerebrospinal rhinorrhea following **ethmoidectomy**.

Case Report.—M. F., female, 35 years of age, was admitted to the medical department of the University of Pennsylvania Hospital on January 20, 1926, complaining of frequent attacks of asthma.

The history of her present illness showed that she had been well until three years ago, when she suddenly developed a severe attack of asthma in the fall of the year. In the following spring she had another attack, and since then the seizures have become more frequent and usually have occurred at night. The attacks began with a stuffy feeling of the head, cough, shortness of breath, watering of the eyes and profuse secretion from the nose. These symptoms were followed in a short time by difficult wheezing respiration, severe coughing and later by shaking chills. The cough was productive of a white tenacious phlegm and she occasionally vomited.

Urticaria sometimes occurred. These asthmatic spells seemed to come on without relation to weather or food. There were no plants in her home, but the family kept a dog and there were chickens in her neighborhood. She was not sensitive to dust.

During the four weeks before admittance to the hospital she had a dozen of these severe attacks. There was no history of chest pains, hemoptysis or night sweats. There were no important gastrointestinal or cardiac symptoms.

The previous medical history showed she had had the ordinary diseases of childhood and also Bright's disease three years previous to admission. She was subject to frequent colds and bronchitis.

Family history: The father was living, at 67 years of age, but had had cardiac asthma for the past twenty years and formerly had frequent attacks of hay fever.

Physical examination: The patient was a fairly well nourished woman of rather cyanotic color. The head, neck, eyes and ears were negative. There were pronounced dental caries and the tonsils were large, embedded and unhealthy in appearance. Examination of the nose was negative except for a mucopurulent discharge in the middle meatus of the right nostril and an enlarged right middle turbinate making firm pressure against the septum. The lungs were negative except for high-pitched rales heard throughout the chest. An X-ray examination of the chest was also negative. Urinalysis showed a trace of albumin but no casts. The kidney function test showed a total elimination of 60 per cent in two hours. X-ray examina-

tion of the sinuses showed the maxillary and sphenoid sinuses to be negative but the frontals and ethmoid cells were hazy.

Skin sensitization tests for chicken feathers, orris root, wheat, dust P., goose feathers, dog hair, oats, sheep's wool, egg, dust G., cottonseed, milk and dust M. showed only a weakly positive reaction to chicken feathers and questionable reactions to orris root, egg and dust proteins. It was decided that these were probably not factors in producing the asthma.

She was transferred to the department of otolaryngology for operation and treatment. It was thought that an operation to relieve the crowded condition in her right nostril and to remove the probably infected ethmoid cells might benefit her asthmatic condition. Therefore, on February 3, 1926, under local anesthesia, the writer removed the right middle turbinate and exenterated the anterior ethmoid cells by the use of punch forceps and curette. The intention was to make a thorough removal of both anterior and posterior cells, but while working on the anterior cells the patient developed such a severe asthmatic attack that it was impossible to continue. There was no packing inserted.

It was noticed while the patient was still in the operating room that there was a rather free discharge of a thin bloody fluid into the throat. On removal to her bed in the ward this discharge became more profuse, soon lost its tinge of blood and became a clear, thin, watery fluid that dripped constantly from the right nostril when the head was forward or ran back into her throat when the head was thrown back. This dripping continued constantly for twenty-four hours following the operation and was so obviously cerebrospinal fluid that no one thought to have it examined until the discharge had stopped and the fluid was lost.

The patient had no symptoms accompanying this discharge except a generalized headache of moderate severity which lasted for several hours after the operation. After consultation with Dr. Fetterolf, it was decided to encourage the flow of cerebrospinal fluid as much as possible in order to constantly wash out any infected material that might be apt to gain entrance to the meninges. To promote the flow the patient was given large amounts of liquid to drink and her head was kept on a level with her body. At the end of about twenty hours

there was some reduction in the amount of the dripping, and in four or five hours more there was practically none.

Following the subsidence of the discharge the patient made an uneventful recovery. The temperature remained normal, and there was no more headache. Her asthmatic attacks decreased from two to four daily during the few days before operation to only three mild attacks during the next two weeks. She was discharged very much improved on February 17, 1926, two weeks after her nasal operation.

We heard nothing of this patient for a month, when she again came to the hospital with the history of having developed a severe attack of grippe soon after her return home. This grippe lasted ten days and precipitated an attack of asthma which was not serious and required no special treatment. She had gained weight somewhat in spite of her illness and felt well but wished to see if anything more could be done to prevent a return of her asthma.

She was readmitted to the hospital on March 17, about six weeks after her operation. Her physical examination was practically the same as on the previous admittance except that the chest was negative for rales or any other evidence of asthma. Examination of her nose showed the presence of two polypi in the right nasal cavity, one hanging down almost to the floor of the nose and apparently attached in the middle meatus, and one in the ethmoid region, with a synechia between the septum and ethmoid region back of the polyps. The left nostril showed a slightly enlarged middle turbinate pressing over against the septum but no evidence of an active ethmoiditis.

On March 24th, under local anesthesia, the left middle turbinate and the two polyps in the right nostril were removed and the synechia broken down.

Following this operation the patient's recovery was slow and eventful. She developed what in all probability was scarlet fever, with sore throat, high fever and a scarlet rash. This was followed in a few days by acute purulent otitis media of the left ear, which required myringotomy. These conditions slowly improved and she was discharged on April 14th, three weeks after her operation.

During the year following her discharge from the hospital she had only two mild attacks of asthma. At no time had there

been any discharge from her nose that in any way suggested a possible return of the dripping of cerebrospinal fluid.

COMMENT.

This case illustrates a rare and serious complication following operation on the ethmoid sinuses. It was expected that the condition would promptly terminate in a meningitis, but no untoward symptoms whatever followed the operation. It would have been interesting and instructive to have thoroughly examined the right ethmoid region while the fluid was dripping to determine the exact conditions present, but this would have meant the cleansing of the blood clots and other material from around the operated area. It was thought better to avoid any intranasal manipulation because of the fear of carrying infection into the meninges, and to let the fluid drain as much as possible in order to carry infected material away from the meninges. The drainage was therefore encouraged by putting the patient on a forced liquid intake and keeping her head low.

Speculation as to the cause of the escape of this fluid leads one to the opinion that in curetting the ethmoid cells a spicule of bone attached to the dura might have been torn away, leaving a small opening in the dura, or there may have been a necrotic process of the bony structure in the deeper ethmoid cells that caused a breaking down of the wall protecting the dura and a partial or complete destruction of the underlying meninges. The fact that this patient recovered, as did also several others of a similar kind reported in the literature, gives one the impression that this complication may not be as serious as is commonly believed.

There is no established treatment for this condition. Practically all authors are agreed that all intranasal manipulation should be strictly avoided. There have been tried adrenalin subcutaneously, atropin internally, sodium iodid internally, thyroid extract and benzyl benzoate, but none of them seem to cause any reduction in the amount of flow from the nose. Restriction of liquids and repeated lumbar puncture will decrease the flow of fluid and is probably indicated in the type of cases accompanied or caused by increased intracranial pressure. However, in postoperative cases it seems questionable whether any attempt should be made to lessen the flow because the

constant passing of the fluid over the injured area will tend to wash any infected material away from the meninges while the reparative process is going on. So long as there is not an increased intracranial pressure it is unlikely that the subarachnoid fluid would be sufficient in pressure to prevent the closure of the perforation through the dura.

#### SUMMARY.

The eight cases herein reviewed are illustrative of the rare complication of cerebrospinal rhinorrhea following intranasal operations. Three died from a cephalomeningitis and five survived with cessation of the discharge. Three cases followed ethmoidectomy, one sphenoethmoidectomy, one the removal of polyps and the middle turbinate, one the treatment of a sinus infection, and one the rasping of a frontal sinus.

The treatment of seven of the eight was conservative, with strict avoidance of intranasal manipulation. In such cases of traumatic origin it probably would be better to encourage drainage rather than to attempt to lessen it. The fact that five of these eight cases recovered shows that this complication, while extremely serious, is not necessarily fatal, as was at one time believed.

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## LXIX.

### PNEUMONIA FOLLOWING EXTERNAL OPERATION ON FRONTAL SINUS: DEATH.

BY ROSS HALL SKILLERN, M. D.,

PHILADELPHIA.

Male, 28. Diagnosis, chronic frontal sinusitis (left). Operation, Lothrop modified. Frontal sinus healthy, anterior superior ethmoid cell extending to the outside of frontal sinus infected. Partition removed, throwing both cavities into one, and diseased mucosa removed. Large counter opening made into nose with special rasp. Rubber catheter drainage. Wound completely closed with silkworm gut sutures.

First day. Normal reaction until 10:30. Temperature went to 102.2; complained of being hot. M. S. by hypo., had good night, slept five hours.

Second day. Temperature down to normal at 11 a. m., symptoms of retention of urine. Catheterized, 23 oz. Slight headache. Nine p. m., temperature 101.2. Pain in back. Had fair night; slept five hours.

Third day. Backache. Temperature 99.4 early morning, but gradually ascending until 7 a. m., reached 105. Up to this time pulse has not been above 90 nor respirations 24. Pulse rises gradually to 120, respirations 40. Lungs examined by internist with no symptoms. Nephritin Tab., gr. 1, every two hours. calomel, gr. iv., sod. bicarbonate enema and entroclysis. Patient perspiring freely and says he feels better, little backache. No pain in chest. No difficulty in breathing. Temperature dropped to 101, but rose almost immediately to 104.1. Patient had poor night; slept four hours at short intervals.

Fourth day. Temperature down to 102, but rises to 105.2, remaining up for nine hours. Sponges every hour hours, no effect. Treatment symptomatic. Patient had fairly good night; slept five and one-half hours; general condition much improved. Voided naturally two good B. Ms.

Fifth day. Wound over eye entirely healed, stitches and drain removed. Temperature down to 101.2 in early morning,

gradually rose to 105.2 by 3 p. m. Patient appears very toxic, as has been the case since second day. Lungs apparently clear. Pulse 120. Respiration continues 24 despite high temperature. Patient had fairly good night, disturbed, however, by cough; slept five hours.

Sixth day. Temperature remains between 102.2 and 104. Coughing more. Examination of lungs showed beginning consolidation of lower right lobe. This has evidently been a central pneumonia, which is just making its appearance on the surface. Sputum sent to laboratory, which showed pure culture pneumococci. Huntoon's antibody solution, 100 cc., given intravenously. Temperature slowly dropped to 102. Patient became slightly irrational during the evening and had poor night.

Seventh day. Temperature rises to 140. Pulse remains around 120; 50 cc. antibody solution administered. Fairly good night. Cough very troublesome, but rested better than the night before.

Eighth day. Temperature, pulse about same, but respiration rises suddenly (within a period of five hours) to 52. Digitora tablets, gr. 1, every fourth hour. Patient resting fairly comfortably, cough not troublesome; small amount of bright blood in expectoration. Does not complain; mind clear. Good night, sleeping six hours; takes nourishment well.

Ninth day. Temperature and respiration same. Pulse 110. 50 cc. antibody solution given. Seems very drowsy; marked difficulty in breathing; slept five hours. B. P. S. 130, D. 60, P. P. 70.

Tenth day. Condition about same, although very cyanotic. At 6 p. m., pulse rose to 130 and remained around 120 to 130. Pulse full. Very apathetic; would rouse and converse but preferred to remain quiet.

Eleventh day. Temperature dropped by crisis from 103.1 to 100.2 within five hours. Pulse fell from 136 to 104. Respiration from 64 to 48. Clear mentally; fine, restful night, according to patient. Had little pain around left diaphragmatic attachment; no rales; breath sounds apparently normal in left lung. Skin dry but cyanotic in face and nails. Within one hour temperature rose to 103.2, pulse 132, respiration 60, with marked delirium and restlessness, requiring restraining by sheet from then until 12 midnight. Temperature remained around

103; pulse steadily rose to 156; respiration to 72. Mentally clear at 11:30, but complained of rales in throat; extreme weakness. Examination of chest showed an apparent acute consolidation of whole left lower lobe. It was assumed that he had developed another infectious process in this area. At this turn his expectoration was scant and watery rather than tenacious and yellow. No blood. Gradually growing weaker; died at 12 M. N. It was noted throughout the day that the abdomen was extremely distended with gas, which condition was not improved as previously with sod. bicarb. enema.

Diagnosis: General pneumococcemia.

Fourth day laboratory report: Pneumococci, types 1 and 4, but stained smear suggested presence of type 3 pneumococcus (in pure culture), because of the heavily stained capsules (Kolmer).

Criticism.—Carefully reviewing this case, I cannot see where any different treatment would have availed. We had, to my mind, a severe generalized pneumococcic infection, probably let loose during the operation, although no culture of the sinus secretion was made. When I again meet with a similar appearing sinus I shall immediately give two intravenous doses of Huntoon's antibody solution, each 50 cc., as a prophylactic the day of and following operation.

ROSS HALL SKILLERN, Operator.

LXX.

EXTENSIVE OSTEOMYELITIS OF FRONTAL REGIONS: MULTIPLE OPERATIONS—DEATH.

BY ROSS HALL SKILLERN, M. D.,

PHILADELPHIA.

Boy, age 16, advanced case of double hyperplastic ethmoiditis. X-ray showed maxillary sinuses both sides and bases of frontal sinus slightly involved.

January 12th. Intranasal exenteration of ethmoid on right. Left hospital in 48 hours. Returned three days later with acute swelling over ascending process superior maxilla on right, which finally ruptured, discharging a considerable quantity of thick yellow pus. Instead of abating, the discharge continued profusely, swelling extending across bridge of nose.

January 31st. Under gas anesthesia, fistula opened and explored, finding that it led directly to the frontal sinus at the junction of the anterior and inferior wall. Drainage established. Despite this, the swelling spread over the bridge of the nose with a pointing to an abscess on the left side identical with that on the right. This was found to lead into the left frontal sinus. As the boy was in the throes of a very acute virulent infection, with extreme suffering and high temperature, no radical procedure was contraindicated. Profuse drainage occurred not only through the external openings but also into the nose, the septum of which soon became the seat of a large abscess, which completely occluded nasal respiration. This was, of course, opened as soon as fluctuation occurred.

A stormy convalescence of six weeks followed, during which time another abscess of the septum, together with acute otitis media occurred. It was necessary to keep the boy pretty well under the influence of morphia to alleviate his sufferings. At the end of six weeks he showed so much improvement it was thought advisable to send him home to recuperate, as he lived in the country, although both fistulae alongside of the nose were still discharging pus which had become thinner and lost its yellow appearance. The patient, however, had lost consid-

erable weight and was in no condition to withstand a radical operation. After being at home three months he returned in fine condition, having regained his former weight and strength. The fistulæ, however, were discharging about as usual.

On April 29th, I performed a double, external, radical frontal (Lothrop method), and found two large sinuses extending outward almost to the end of the eyebrow and upward about the same distance. These were filled with a thickened, bluish, discolored pyogenic membrane with pockets of pus at the extremities. Both of the anterior sinus walls were completely removed, with the exception of an oval section on the median line extending outward from the bridge of the nose, which was left for cosmetic purposes. The lower part of the intranasal septum was completely removed, together with the upper part of the nasal septum. Also the anterior superior nasal spine was chiseled away, converting the two openings into one, large enough to insert one's index finger. Rubber catheters for drainage were placed in each sinus and cut off at the tip of the nose. The periosteum was drawn together and sutured with fine catgut, then the skin incision closed with fine silk-worm gut and taped with iodoform gauze laid over the incisions with superlying gauze and fine bandage.

Convalescence about as usual. Stitches partially removed on the fourth day and completed on the sixth. Left fistula completely healed, right fistula again broke down and began to discharge. Examination revealed bare bone denuded of periosteum at lower portion of opening (ascending process of superior maxillary). Opening packed with iodoform gauze. Fistula gradually closing, although still discharging some thinner pus. Edges touched with lunar caustic (Ag. No. 3). Three days later still discharging, although smaller. Filled with bismuth paste. Still considerable purulent discharge from right side of nose, although abating daily. Treatment discontinued.

Patient returned in two months with acute swelling over left sinus, including periorbital tissues. Under local treatment, soon returned to normal. Patient again returned home, and in about two weeks suddenly developed extradural abscess on left side, to which he succumbed.

Criticism.—I feel had I this case to treat over again I would have made the incision much earlier into the swelling below the

eye alongside of nose on the right side in order to determine where this collection of pus was having its origin. In this way, I believe that by freely draining the right sinus externally at this stage it might have prevented the left one from becoming infected, as well as the nasal septum and middle ear. Whether this would have removed the possibility of an external operation, no one, of course, can say. Regarding the sudden appearance of an extradural abscess, this was not surprising, as the frontonasal bones were pretty thoroughly involved in this low grade form of osteomyelitis, but even so would have offered a good chance for recovery had an early operation been performed. This was, however, impossible, as his home town was not quickly accessible.

LXXI.

EXTERNAL OPERATION ON THE MAXILLARY  
SINUS (CALDWELL-LUC), FOLLOWED  
BY DEATH.

BY ROSS HALL SKILLERN, M. D.,

PHILADELPHIA.

James G., male, age 58, came to us October 22, 1927, with the following history: Eighteen months ago he began to have pain over the right maxillary sinus and through the right supraorbital region, with obstructed breathing on the same side. He stated he was operated on intranasally in October, 1926, and notwithstanding the operation, his condition progressively grew worse.

Rhinoscopic examination revealed a profuse discharge of pus coming from under the anterior half of the right middle turbinate, which by lavage through an accessory ostium was proven to be from the right antrum. On the day of his examination he showed evidence of dejection and a general picture of decrepitude. It was obvious that he was a poor operative risk; nevertheless, on account of his septic condition, operation was deemed advisable and a Caldwell-Luc was performed on October 24th. Anesthesia: Nitrous oxid, oxygen, ether sequence.

The operation proceeded as usual, no difficulties being encountered and very little bleeding, the entire time requiring forty minutes. The patient was covered with warm blankets and placed immediately in bed.

Operative Findings.—The maxillary sinus was filled with thick yellowish green pus, some odor. The entire mucous membrane lining the cavity was of a dark bluish red color, markedly thickened and polypoid in character, a considerable area being loosened from the underlying bone. Immediately following the operation he suffered some shock, which was not so profound, but quite prolonged. Temperature did not fall below 98 or pulse above 120. He was given hypodermo-

clysis, strychnin sulphate, caffein citrate, tincture digitalis and heat applied externally. On the second day after the operation his condition appeared to be improved, only to be followed on the next day by untoward and eventually fatal symptoms, death occurring on the third postoperative day at 8:10 p. m. Autopsy urged but not permitted.

Criticism.—This patient was markedly septic, evidently from the old chronic suppurative process in his right antrum. He had also lost considerable weight and strength in the last three months, the cause of which had been undetermined (certainly not tubercular). The operative prognosis was not particularly encouraging, but considering the difficulty in making a successful needle puncture and the unreliability of cleansing the sinus through the middle nasal fossa, together with the rapid progress of the patient's exhaustion, it was thought the best hope lay in the immediate exacuation of the purulent secretion with removal of the pyogenic membrane, followed by aeration and drainage.

If we had this problem again to face, it is difficult to see how we could have well done otherwise. While the patient gave his age as fifty-eight, he appeared to be nearer seventy. To have tried conservative measures would have been futile, as permanent pathologic changes had taken place in the mucosa, absolutely precluding the possibility of returning to normal, and the only hope of a real cure lay in the bone forceps and curette.



LXXII.

INFREQUENT ANGINAS: DESIRABILITY OF  
THOROUGH LABORATORY STUDY.\*

By V. K. HART, M. D.,

STATESVILLE, N. C.

The above subject may seem to portend little importance. It is hoped quite the contrary will be shown. Moreover, common conditions, such as acute follicular tonsillitis, diphtheria, secondary syphilis, Vincent's angina and scarlet fever will not be discussed. Such problems are well understood and the therapy well standardized. Much more attention is merited by infrequent anginas—that is, those not coming under one of the above groups.

Thus, a complete blood count, during an angina of obscure origin, is just as important as smear and culture. In the first place, one cannot tell with certainty but that the same is a beginning agranulocytic angina. The author has recently reported one such case<sup>5</sup> and has more recently had another under observation.<sup>6</sup>

Perhaps it would be better in these cases to say "septicemia with low white count," inasmuch as both cases had positive blood cultures. Moreover, Kastlin,<sup>7</sup> in a splendid article on this interesting subject, shows that very possibly agranulocytic angina is a distinct clinical entity without true sepsis and one to be differentiated from a septicemia with low count. He describes ulcerated and necrotic areas in the oral cavity. The truth of the foregoing remains to be more fully exemplified. All the more argument for routine counts and microbic study in these acute throats. Certainly laryngologists should take the initiative in working out the problem and not leave the same wholly to the realm of the internist.

There is still another rôle of the blood count in acute throats, viz., in so-called infectious mononucleosis. This malady often begins with an acute throat and generally some cervical adeno-

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\*From the Department of Head Specialists, Davis Hospital.

pathy. The name, as Cottrell points out, is very unfortunate, because the increase is not in the endothelial cells but in the lymphocytes. A better name, and one sometimes used, is "acute lymphadenosis."

These cases heretofore have often been diagnosed acute lymphatic leukemia. Indeed, the writer saw one such case following tonsillectomy, with widespread adenopathy and marked relative lymphocytosis (80 to 90 per cent), in which he concurred in such a diagnosis. The subsequent recovery and continued health proved unquestionably the case to be one of so-called infectious mononucleosis.

The prognosis of the latter is distinctly favorable. That of acute lymphatic leukemia, however, is distinctly unfavorable. Hence the importance of an accurate initial diagnosis.

The outlook in agranulocytic angina is not good and is characterized by a marked, persistent low white count, the diminution being largely in the granular leukocytes or polymorphonuclears. The blood picture of infectious mononucleosis is not always so easily recognized, but frequently repeated counts will usually determine the diagnosis. In acute leukemia the white count is usually higher (not always, as in aleukemic leukemia), the hemoglobin and red cells much reduced, with a low color index and general weakness more marked.

A typical and interesting case of infectious mononucleosis recently observed by the writer is cited below. She presented herself for examination on September 24, 1927.

History—C. C.: Swelling of the eyelids. H. P. I.: For past week has had intermittent puffiness of the upper lids with some malaise. No other complaints of note. P. M. H.: Essentially negative. F. H.: Negative.

Examination—General: Well nourished female, age 29 years. Eyes, marked puffiness of upper lids, which is symmetrical. Lower lids not involved. Rest of eye examination negative. Ears negative. Nose negative. Throat, some slight reddening of fauces and throat (curiously enough, she did not then complain of sore throat, but did the next day and for several days thereafter). There was a moderate adenopathy of anterior triangles of neck.

The rest of the physical examination was entirely negative. Such included a fluoroscopy of the chest.

Temperature, 100.6. Blood pressure, 108/72.

Laboratory: Urinalysis negative. W. B. C., 19,800; polymorphonuclears, 13 per cent; lymphocytes, 87 per cent; hemoglobin, 72 per cent.

Culture of throat staphylococcus albus. Smear from throat negative for Vincent's organisms.

Tentative diagnosis: Infectious mononucleosis with possibility of acute lymphatic leukemia.

#### SUBSEQUENT BLOOD COUNTS.

|          | Hb. | R.B.C.    | W.B.C. | Polys. | Lympha | Mono. | Baso. | Eosino |
|----------|-----|-----------|--------|--------|--------|-------|-------|--------|
| Sept. 26 | ... | .....     | 33,500 | 15     | 85     | ....  | ....  | ....   |
| Sept. 27 | 78  | 5,450,000 | 24,700 | 13     | 86     | ....  | ....  | ....   |
| Sept. 29 | ... | .....     | 24,300 | 21     | 79     | ....  | ....  | ....   |
| Oct. 1   | 78  | .....     | 12,500 | 21     | 79     | ....  | ....  | ....   |
| Oct. 2   | 75  | 4,290,000 | 10,150 | 20     | 78     | ....  | 1     | 1      |
| Oct. 4   | 78  | 4,550,000 | 9,600  | 26     | 69     | ....  | ....  | 5      |
| Oct. 7   | 78  | 4,590,000 | 5,800  | 33     | 65     | ....  | ....  | 2      |
| Oct. 15  | 78  | 4,040,000 | 7,450  | 45     | 53     | 2     | ....  | ....   |
| Oct. 20  | ... | .....     | 9,050  | 37     | 59     | 2     | 1     | 1      |

Clinical Course: She ran a temperature ranging from 100 to 102 for one week. The throat remained injected and sore for four or five days. There were no exudative, ulcerated or necrotic areas. There was some malaise, but the patient was never prostrated. Otherwise there were no findings of note.

After the first week she made an uneventful convalescence. She was married October 22nd and left the community, but reports from her family indicate she has remained well.

Comment: An accurate prognosis in the above case was certainly of the utmost importance in view of her approaching marriage. That the diagnosis of infectious mononucleosis was correct is proved by her rapid improvement and her subsequent good health. Certainly a diagnosis of leukemia would have been a grave error.

The histology of a tonsil and lymph node removed during the course of the disease is not distinctive.<sup>3</sup> The reported slides do not show organisms. It would appear then, that the lymphatic system is probably affected by a toxemia with a certain specific type of infection yet to be determined, predominating in the throat.

The etiology of infectious mononucleosis is then obscure as is the *modus operandi*. Is it merely a toxemia from any acute throat? Is it a toxemia from one and only one type of

infection in the throat? Is it primarily an infection of the lymphatic system with the throat as a portal of entry?

Several pertinent questions may also be asked about agranulocytic angina. Is it a primary throat infection with overwhelming toxic paralysis of blood forming organs? Is it a blood stream infection with the same result and the throat as a portal of entry? Or is the marked diminution of polymorphonuclears merely a secondary condition which may result from either of the above conditions? If the first, is the primary throat infection always the same organism? If so, what is the organism?

The two recent cases observed by the writer both had positive blood cultures (streptococcus in the first and Friedlander's bacillus in the second). Both had very acute throats but no ulcerated or necrotic areas of the buccal cavity, as usually described in agranulocytic angina.

Hence it would seem that sepsis with a low white count, indicating very low resistance or a very virulent infection, constitutes one group of cases. Another group comprises those with necrotic and ulcerated lesions of the buccal cavity without a positive blood culture. The writer has a feeling that such oral lesions are due to some systemic disease and not necessarily to some undiscovered bacterial infection. Agranulocytic angina is yet to be proved a definite, clinical entity rather than purely a secondary condition.

Digression is made for a moment to epidemic, acute throats of neither diphtheritic nor scarlatinal origin. One such epidemic recently occurring in a girls' college was observed. Twenty-one cultures were mixed. Of these, nonhemolytic streptococcus predominated in twelve; staphylococcus albus in nine. Five other cultures were staphylococcus albus and one was sterile. Smear from the twenty-eighth patient showed Vincent's organisms.

These patients all became ill in the same few days. A moderate elevation of temperature was usually present and some malaise. Uneventful convalescence occurred in all cases. Unfortunately, blood counts were not made on this group, but none showed any unusual lymphatic disturbances. The milk supply was suspected, but careful bacteriologic study of the

same failed to show pathogenic organisms. Streptococcus in particular was suspected.

A number of cases of fungus infections of the oral cavity have been reported. Such should always be borne in mind in unusual lesions.

There is still another condition which deserves passing mention among unusual lesions of the throat, because it sometimes first manifests itself in the mouth or throat—i. e., pemphigus chronica or vulgaris. It is usually fatal, and the etiology has not been solved. In a case reported by Fox<sup>4</sup> there was little alteration in the blood count, there being a slight leukocytosis only.

#### SUMMARY.

1. Agranulocytic angina is yet to be proved a definite clinical entity.
2. In unusual anginas, particularly with adenopathy, one should always think of infectious mononucleosis.
3. Nonspecific streptococcic throats are interesting clinically.
4. All unusual anginas should be studied thoroughly from a laboratory standpoint by laryngologists. Blood counts in suspected infectious mononucleosis and blood cultures in agranulocytic angina are particularly important.

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LXXIII.

DIFFICULTIES IN DIAGNOSING LATERAL SINUS  
THROMBOSIS.\*

BY NATHAN P. STAUFFER, M. D.,

PHILADELPHIA.

This paper is to illustrate some of the difficulties in determining the certainty of a patient having a lateral sinus thrombosis. The work of Dr. B. F. Crowe (Baltimore), published in a pamphlet I saw him preparing in Vienna in 1912, gave us a working basis. In this he recommended compressing the opposite jugular vein from the suspected mastoid and sinus and if a clot was present he expected to see the veins of the forehead very much engorged, and this was supposed to be conclusive evidence of a lateral sinus thrombosis. But the engorgement of the veins did not always appear—whether from faulty pressure, or some other reason I do not know. Lately the manometer has been considered more reliable. In this paper I will record two cases where the manometer proved, in one, of invaluable help and in the second case contradicted our findings.

March 8, 1927. V. P., white, female, aged 16 years, student at Westown School. Patient of Dr. Roberts, Lansdowne, with a history of spontaneous rupture of right ear drum and profuse discharge and left ear ac. otitis media. Three days later left ear had a paracentesis under local. March 18, 1927, patient was admitted to Presbyterian Hospital with temperature 100 F., pulse 88, W. B. C. 11,000, hemoglobin 50 per cent. Mastoid tenderness, persistent headache. Dr. Eves and I did a mastoidectomy on March 18, 1927, which did not present any unusual symptoms, except headache. Three days later her temperature rose to 103, only to return to normal range three days later. It remained between 98 and 100 for a week, during which she complained of an increasing headache, having a temperature of 103, and one slight chill. Consultations with Drs.

\*Read before the Section of Otology, College of Physicians of Philadelphia, September 19, 1927.

Eves, S. MacCuen, Smith, Roberts and Talley, were held to differentiate between lateral sinus and meningitis. Dr. Thorington reported negative eye ground changes, so Dr. Eves and I agreed to have Dr. Eiman do a spinal puncture, as the consultants could not agree on the diagnosis. Spinal fluid clear. At this time we were in doubt whether she had a sinus thrombosis or a meningitis, as she did not have any of the typical septic chills accompanying a clot. Pressure was made by Dr. Eves on the jugular vein on the suspected lateral sinus side, with manometer ( $H_2O$ ) in position while Dr. Eiman did a spinal puncture. The pressure was only 250, while pressure on the free jugular vein sent the pressure gauge to 450. We concluded we had a thrombosis and opened the lateral sinus and removed the clot, which extended as far back to the torcular and as the probe would reach. A fairly good flow of blood followed, but none from the jugular end. The wound was packed with iodoform and the jugular ligated but not resected. In spite of this the temperature remained around 102, 103 and 104, with persistent headache for the next twelve days. We used every means to locate the cause of the temperature and headaches. Realizing we had a very sick child on our hands, Dr. Eagleton was called in consultation, and we debated the advisability of ligating the other jugular, as there was a probability of the clot having extended to the other side of the occiput. We decided to give her a blood transfusion on account of her marked anemia. Dr. John Speese gave her 400 C. C. of blood, from a resident which was immediately followed by a marked amelioration of her symptoms. A week later she was given a second transfusion which promptly cured her. She left the hospital ten days later and has had a complete recovery.

The second patient, M. S., white, female, aged 15 years, a patient of Dr. Hargett. Admitted to Presbyterian hospital, September 17, 1927. History of bilateral chronic purulent otitis media since one year of age, following scarlet fever and a diagnosis by her M. D. of probable meningitis. She had been taken home from school three days previous to admission, suffering with headache, nausea, vomiting, and dizziness. Examination revealed a small amount of a foul discharge from attic, both ears, with destruction of membrana tympani. Tem-

perature 102, pulse 120, respiration 20, R. B. C. 3,500,000, hemoglobin 60, W. B. C. 12,400. Dr. Cleland—spinal puncture, mercury; pressure 8; clear fluid. No mastoid tenderness, or swelling, no drooping of the posterior wall. No nystagmus. Hearing—very deaf. We were in doubt whether we were dealing with a meningitis as suggested by her appearance and her drowsiness; or a brain abscess, or some abdominal lesion as she never had a normal bowel movement. Dr. Langdon reported negative eye grounds. Dr. Eiman performed a lumbar puncture and the fluid was clear—pressure 170 mm.  $H_2O$ . Compression of the jugulars gave a rise of 350 to 400 (each side), no marked difference. Dr. Eiman and I concluded she had no thrombosis. In view of her serious condition, a radical mastoid was performed in which I was assisted by Dr. MacFarland and Dr. Tate. The lateral sinus was found uncovered during this operation and its wall was covered with granulations, but on pressure it seemed to fill up again. The radical operation was completed and the wound was about to be closed when I noticed a small drop of pus appearing on the sinus. I decided notwithstanding the manometer being negative and the sinus apparently filling after pressure, I would open it. I found the lateral sinus filled with a large clot and pus—it was cleaned and packed as usual. My patient did very well for five days when her temperature again ascended to 104 F. on September 24, 1927, and her headache returned with a chill. We had about decided to resect the jugular vein when she suddenly complained of sharp pains in her left chest area. Dr. Talley reported negative lungs, but a slight pleurisy and strapped her side. We decided to ligate and resect the jugular when she had two chills and continued fever.

Dr. Kline and Dr. Town on September 27, 1927, performed the usual jugular vein resection. She did not improve as we hoped and had a temperature every day with chilly sensations. Remembering my success with the patient in June, I asked Dr. Speese to give her a blood transfusion; 500 c. c. from one of her family brought her temperature and condition back to normal.

Case 3. A. J. D. A., white, female, aged 10 years, patient of Dr. Hancock. Admitted to the hospital with acute purulent otitis media and temperature 104, pulse 152, W. B. C. 19,850.



An immediate mastoidectomy was performed. The following day the temperature again rose to 104 and remained in that region for the next twelve days. Consultations were held with several eminent otologists, but we could not agree as to the cause as she did not have any definite symptoms except a slight headache and her high temperature. Lumbar puncture showed a clear fluid. Compression of jugulars was negative. She then developed a pain in the left side of her chest and her eye became sore and the conjunctiva deeply injected.

July 3, 1920, Dr. Radcliffe saw her in consultation, because the eye grew worse, sclera was deeply injected and conjunctivitis worse, but he said nothing in eye grounds to account for her temperature. July 12, 1920, transillumination—good pupillary reflexes and no shadows, no pus in left nares or right. Tonsils O. K.

This case illustrates how a supposed pleurisy, or a possible sinusitis symptom with eye trouble can confuse us as to a lateral sinus thrombosis, also how it is possible to have a septic blood clot, sufficient to cause high temperature, with negative blood findings.

On none of these days did she have the typical septic sinus rise of temperature, or any chill. On the thirteenth day she had a wide excursion of the temperature and I decided to open her lateral sinus even though it had been unexposed at the operation. She had a large clot and it was evacuated and a free flow of blood was secured from each end, culture from blood clot and pus in sinus showed streptococci hemolyticus. In spite of this her temperature continued high and she had chills; blood culture negative. However, she gradually returned to normal.

Case 4.—D. S., white, female, aged 10 years, patient of Dr. Henry Jump, acute mastoiditis following measles. Admitted to hospital with temperature 102. Immediate simple mastoid operation performed and streptococci found in the pus. After the operation her temperature kept rising until it read 106 F. Her wound was opened and no pus found. She complained of abdominal pains but no localized tenderness. Two days following operation she had a chill. W. B. C. count was now 20,300, and blood cultures showed the same streptococci we found in the wound. She had some vomiting, so we decided

to open her lateral sinus and found a large thrombosis. Her temperature dropped to 97 and the following day rose to 106 and we gave her 500 c. c. of antistreptococci serum as Dr. Eiman reported a bacteremia. These brought down her W. B. C. to 16,000, but her temperature kept rising. Two days after the first operation we ligated and resected her jugular vein. Still her temperature kept rising till it reached 106  $\frac{2}{5}$ , when she complained of pain in her leg. Dr. Jopson diagnosed and opened an abscess in her right leg, still her temperature kept rising each day, until it reached 108 F., when she had pain in her left leg. It remained between 106 and 108 for two weeks during which she had abscesses in both arms and back of her neck. She then recovered. She is now earning her living, many years later.

Case 5.—S. M., white, male, aged 10 years. Admitted to hospital with temperature 104 to 105 F., with history of discharging ear for four days. Following observation—for several days, a mastoid operation was performed. The following day his temperature rose to 105 F., with daily excursions from 101 to 105 and we were greatly worried as to the cause of this high temperature. We were in doubt whether we had a lateral sinus thrombosis, a labyrinthitis, or a meningitis. Three days after operation he had a chill, but no evidence beyond that of a thrombosis. Pain in right leg developed next day, and later extended to his knee and then to his thigh. He evidently had a metastatic thrombosis in his right leg, which ultimately recovered under a plaster cast and rest.

Case 6.—K. M., white, female, aged 7 years, patient of Dr. Service, admitted to the hospital with acute purulent otitis media, following tonsillitis; W. B. C. 24,400, temperature 100, pulse 128. X-ray reported mastoiditis. Under gas oxygen anesthesia a simple mastoid on right side was performed. Following this her temperature remained around 100 F. to 102 F. and the child became very irritable and complained bitterly and constantly of headaches. We were in doubt whether we had a brain complication or a lateral sinus thrombosis. I was surprised one morning to notice her right eye did not rotate externally. In order to eliminate meningitis we had Dr. Cadwalader go over her, and he believed her sixth nerve paralysis was due to an inflammation of the petrous portion of her tem-

poral bone without a definite meningitis. Dr. Langdon reported a paralysis of the right (eye) external rectus (the side operated upon) and engorged veins. I am always suspicious of a thrombosis when they have headaches and a temperature following mastoiditis, even though they do not have chills and the pyemic temperature. I had been exhausting every test to ascertain the exact cause of her temperature and headaches. Whether these headaches and temperatures came from the inflammation of her petrous portion I am unable to state positively. I only know that under ice bag and appropriate remedies and rest she made a complete recovery.

These cases illustrate the difficulty of definitely diagnosing a lateral sinus thrombosis until it is quite well established.

#### CONCLUSIONS.

1. The most common and regular symptoms seemed to be headache with special reference to the frontal and occipital regions.

2. The temperature ranges were varied. See case charts.

3. In only one did I see the typical chill and cyanotic appearance, Case No. 4.

5. Only one to have a positive blood culture.

6. Metastatic involvement in other parts of the body may mislead us into a wrong diagnosis of a lateral sinus thrombosis.

7. In each case the streptococcus hemolyticus was the cause.

8. Manometer findings not positive, but of probably great value when standardized.

9. The varying length of time the disease lasted before the disease became involved—No. 1, in one week; No. 2, over 14 years; Nos. 3 and 4, in two weeks; No. 5, in four days, and No. 6, in ten days.

10. High W. B. C. in all cases.

11. These demonstrate how resistant the blood stream itself is to a fatal infection, and show how in spite of being desperately sick you must never lose hope or stop trying.

12. These two cases demonstrate the value of blood transfusions and hereafter I shall give them earlier.

1900 RITTENHOUSE SQUARE.

CEREBRAL HERNIA COMPLICATING CHRONIC  
MASTOIDITIS, PERISINUS ABSCESS, THROM-  
BOSIS OF THE LATERAL SINUS AND  
JUGULAR VEIN; OPERATION  
WITH RECOVERY.

BY HENRY S. WIEDER, A. M., M. D.,

PHILADELPHIA.

In reporting any case that we encounter in our work in otolaryngology, one feels a hesitancy for fear that he is burdening the literature with material which has been written about innumerable times before, only possibly in much better manner. Accordingly the writer feels that his justification for reporting the following case is to bring forward a rather unusual group of complications and to make it a text for discussion of a much mooted question in treatment upon which he has not been able to find any comprehensive review in the literature.

The history of the case is as follows:

T. S., male, age 25 years, gave a history of having had a mastoidectomy performed on the right mastoid bone in 1914 while still in Italy. He has had a chronic discharge since. About a week before admission to the hospital he developed pain and swelling behind the right ear. It was incised and pus evacuated at one of our large hospitals. A foul discharge persisted from behind the ear and through the canal. He was admitted to the Northern Liberties Hospital May 2, 1927, for observation and to have a radical mastoid performed. On admission his temperature was 97.4 and remained around normal for four days, with two rises to 99.4 and 99.6. Urine examination showed albumen, plus 1, otherwise normal. Blood R. B. C. 4,090,000, hemoglobin 80 per cent, leucocytes 10,000, polys 75 per cent, large lymphocytes 5 per cent, small lymphocytes 20 per cent. X-ray study showed the evidence of the

previous mastoidectomy and haziness of the remaining bony structure.

On May 6, 1927, on the morning of the day set to perform the radical mastoidectomy, the patient suddenly developed a violent chill and the temperature rose suddenly to 103.4. A prompt eye, neurologic and medical examination were all negative for intracranial or other medical condition, so that a suspicion of lateral sinus thrombosis was entertained. An immediate blood culture and blood count were taken. The blood showed R. B. C. 3,960,000, hemoglobin 65 per cent, leucocytes 13,000, polys 90 per cent, small lymphocytes 5 per cent, large lymphocytes 5 per cent. At 3 o'clock in the afternoon he developed another chill, and his temperature rose to 105.8. At 5 in the afternoon, as soon as his temperature had dropped to 103, he was taken to the operating room and under ether anesthesia a radical mastoidectomy was performed. Extensive necrosis of the entire mastoid bone was found, with a perisinus abscess leading down toward the bulb. The entire area of the dura was covered with a greenish slough. The lateral sinus was thrombosed and pure pus found within its lumen. The jugular vein was tied and severed between two ligatures low down in the neck, a gauze wick introduced along the vein and the neck wound sutured. The lateral sinus was then opened widely, and a free flow of blood obtained from the torcular end of the vein. It was not possible to obtain a free flow of blood from the bulbar end of the vein. A radical mastoidectomy was performed, and the entire time that the work was being done in the region of the middle ear the patient's face twitched with every introduction of an instrument. The patient stood the operation quite well, although at times was pulseless and needed stimulation upon the table. Culture from blood before operation yielded short chain streptococci, and from the wound and the lateral sinus staphylococci and short strain streptococci.

The temperature promptly dropped down to normal and the patient felt comfortable. He was given daily injections of 20 cc. of Pregl's iodine intravenously. Eye examination the day after operation showed a slight blurring of the disc on the nasal side in the right eye. The temperature remained normal for five days with the exception of two temporary rises to the

neighborhood of 100 degrees. A blood culture taken three days after the operation and all subsequent blood cultures were negative. On the sixth day following operation, May 12, 1927, the temperature rose again without a chill to 104 and hovered around that figure for three days. Eye examination at this time showed some optic neuritis on both sides, worse on the right. The only neurologic symptom of note was a definite weakness of the left facial nerve (contralateral palsy). The neck wound was healed and the mastoid wound looked clean and devoid of odor, but there was some bulging in the lower posterior angle of the wound. He looked rather toxic. Leucocytes rose to 14,000 with 83 per cent polys. On the third day of the temperature rise, there was an excess of discharge upon the dressings, which came from the lower anterior portion of the wound, just in front of the bulging, which was increasing in amount. The temperature promptly dropped and ran a rather normal course for four days, when it again rose to 104 with the appearance of some induration immediately below the mastoid. The following day this induration extended down the neck along the jugular vein, so under local anesthesia a wide opening was made in the neck, liberating about an ounce of foul pus. The pocket extended down nearly to the top of the mediastinum and well up the neck along the jugular vein, which had evidently been filled with a broken down clot, for on washing the wound the next day fluid introduced in the neck oozed out of the mastoid wound. The temperature again dropped and never again rose previous to the time of his discharge. He never had any further untoward symptoms, but the swelling in the mastoid wound continued to increase until it was flush with the surface, on May 31, 1927, and above the level of the wound on discharge June 8, 1927. On May 31, a Bárány caloric test was made on the left or unaffected ear. In a minute and a half, water at a temperature of 60 degrees produced a *horizontal* nystagmus to the left with large slow excursions when the head was erect (perverted nystagmus) and the same nystagmus on putting the head back. The right ear was not tested at this time. Physically he felt perfect, had no headache, could walk well, but owing to the persistence of bulging, which was increasing instead of decreasing, and because of the persistence of the optic neuritis it was determined

that a careful observation should be maintained with continued study, especially with a view to there being an intracranial abscess present without symptoms because of the local decompression and herniation of the brain. The facial palsy had recovered completely.

The patient was discharged to the outpatient department on June 8th, feeling perfectly well, but with a protruding mass in the mastoid wound, which rapidly attained the size of a walnut, was soft, apparently covered with granulation tissue but not pulsating. It could be brought flush with the surface of the bone by pressure but would promptly regain its former size on release of pressure. Pressure on the mass caused no headache, nystagmus or vertigo—in fact, caused the patient no inconvenience whatever. The entire radical mastoid cavity was filled with the mass and also the external auditory canal at a depth of about one-half inch.

On June 25th a complete Bárány test was made at the University of Pennsylvania Bárány Clinic by Dr. Myron A. Zacks, who found spontaneous nystagmus to the right, left and upward on extreme movement of the eyes in those directions. His station was fair, Romberg negative and there was no spontaneous past pointing. Hearing was acute with the left ear and good with the right ear through the dressings.

On turning to the right ten turns in twenty seconds, he developed nystagmus to the left of good amplitude for twenty-two seconds and had vertigo for twenty seconds. Ten turns in twenty seconds to the left developed nystagmic movements to the right for twenty seconds and vertigo lasted fifteen seconds. On turning to the right ten turns in ten seconds he past pointed with both hands to the right, that with the left hands being less in amount and not repeated after the first pointing. Ten turns to the left in ten seconds caused the right hands to point four inches to the *right* while the left touched.

Douching the right ear with bichlorid solution at 68 degrees for four minutes produced no nystagmus with the head up and very poor horizontal nystagmus to the left with the head back. The right hands past pointed two inches to the right, the left hand touched. The response was the same with the head erect and the head back.

Douching the left ear for three minutes with water at 68 degrees produced practically no nystagmus (only a few rolls) and with the head back very good nystagmus *obliquely* to the right. He touched with both hands. The entire test caused no nausea and was completed at one sitting. In the opinion of Dr. Zacks, the findings were indicative of a lesion in the posterior fossa, subtentorial in location.

The writer felt morally sure that the mastoid mass was a cerebral or cerebellar hernia but has never in his experience encountered one after mastoidectomy, so sought the advice of Dr. Temple Fay because of his experience in neurosurgery. Dr. Fay also was unable to give a definite opinion as to the nature of the mass and advised making a section for pathologic examination to determine definitely its nature, claiming that if it is brain structure the herniated portion would be functionless so that no harm could be done. As it was desirable to get a photograph of the mass before interfering with it, section was deferred until one should be obtained. In the meanwhile the surface was painted with 40 per cent formalin, upon the advice of Dr. Fay, and when a crust had formed it was cut away. This had been done several times and the size of the mass considerably reduced, when on July 18th, after cutting off a crust, there was a small amount of bleeding that was extremely difficult to control, and, when controlled, was followed by a trickle of a clear, limpid, colorless fluid, quite evidently cerebrospinal fluid. A tight bandage was applied and the patient sent home, but on his return to the hospital the dressings were found to be soaked with a clear liquid. Accordingly he was readmitted to the hospital and seen again in consultation with Dr. Fay.

It was found that the cerebrospinal fluid leak occurred from the top of the mass and that packing tightly with iodoform against the bony edges of the wound controlled the leak entirely. He was placed on a dry diet with free purgation to reduce the cerebrospinal fluid.

Four days later, July 25th, Dr. Fay, assisted by the writer and Dr. Campus, the resident physician, carefully undermined the skin surrounding the wound, then gradually released the packing which had been introduced four days before, freshened



the skin edges and brought the edges of the wound together with interrupted subcuticular silk sutures and then a continuous silk cuticular suture. All bleeding points were tied with silk. Local anesthesia with novocain, 2 per cent, was used. The wound edges approximated but under great tension.

The patient stood the operation very well. The dry diet with purgation was continued to keep the brain volume down, and thereby keep the tension on the wound down to the lowest level. The wound did not heal by first intention, breaking down somewhat in a few days, but over 50 per cent gain in reduction in size of the wound and in size of the hernia was attained. He was again discharged to the outpatient department on August 2, 1927, feeling perfectly well, with his hernia decreasing in size and gradually being covered over with epithelium. Ever since drainage from the ear became obstructed by the hernia, there has been a constant moisture present, caused by foul smelling thin pus. On one occasion an effort to clean out the external auditory canal a little deeper than usual was followed by a trickle of cerebrospinal fluid that lasted for several days, but eventually ceased under packing the canal tightly with bismuth formic iodid powder and making pressure with a cotton plug and a tight bandage.

The present condition of the patient is that he is physically in perfect condition. He has been working when he has been able to obtain work. His choked discs have entirely disappeared, leaving only the slight optic atrophy, his vision being practically normal. He does not present a neurologic symptom. His mastoid wound is healed posteriorly over the hernia cerebri.

#### COMMENT.

This case presents a number of points of interest, as follows:

1. The presence of such severe pathology and infection with so few clinical or laboratory signs prior to the stormy outbreak of symptoms on the day of operation.
2. The fortunate escape from mediastinal infection despite the fact that the abscess in the neck reached almost to the upper border of the clavicle.
3. The appearance of contralateral facial paresis.

4. The presence of double choked disc appearing after the operation and persisting for several months and the appearance of a cerebral hernia without other signs of intracranial pressure, such as headache, vomiting, nystagmus, etc., and apparently without the presence of a brain tumor or abscess.

5. The explanation for the appearance of a cerebral hernia without an apparent wound in the dura.

6. The point of origin of the cerebrospinal fluid leak.

7. Discussion of the treatment of postoperative brain hernia in infective cases generally and this case in particular.

We will consider the various points in the order mentioned.

To what can we attribute the few symptoms exhibited until immediately before his operation? The condition had evidently existed for some time because the dura was green and necrotic, and pure yellow pus had taken the place of part of the thrombus in the lateral sinus. In addition, the clot had evidently already involved the jugular bulb, since it was impossible to establish a free flow of blood from the bulbar end of the sinus. The condition in the ear was of long standing, and the necrosis had probably been progressing so slowly, meanwhile throwing out protective adhesions in the soft parts as it progressed, that there was very little absorption from the infected area, hence very few clinical symptoms or laboratory indications of the gravity of the condition. The opening of the subperiosteal abscess may have released tension on the pus under pressure and assisted in masking the true condition. When, however, the thrombus broke down and a few bacteria gained access to the circulation, the condition became manifest. This one phase of the case alone is surely evidence of the fact that there is no group of symptoms always present in every case of thrombosis of the lateral sinus or perisinus abscess, but that, in some cases, any or all of them may be absent.

With reference to the abscess in the neck, a possible reason the infection did not run down into the mediastinum was because the jugular vein had been severed completely between two ligatures so that when the infection traveled down the distal portion of the vein already a protective barrier of reparative inflammatory tissue had been thrown around the lower ligature and injured portion of the vein. Had the vein been

continuous so the inflammation could have traveled down the sheath of the vessel, I fear the outcome may not have been so favorable despite prompt opening of the abscess.

The explanation for the contralateral facial paresis has been most difficult. Were this the sixth nerve, the occurrence would not be so unique or difficult of explanation, but how to connect an isolated seventh nerve paresis with an infection of the opposite ear has baffled all to whom it was described. The explanation offered by Dr. Yaskin, who has so kindly studied the case throughout with me, is that it was merely a coincidental peripheral refrigerant palsy.

The double choked disc has been a source of more worry than almost any other feature of the case, for it has kept us constantly anxious lest we may be still dealing with a latent abscess of the brain despite the patient's favorable condition in other respects. When in addition, the hernia cerebri presented itself, both conditions persisting over a long period of time despite amelioration of all other symptoms, and finally when the Bárány findings late in the case indicated some intracranial disturbance, our uneasiness appeared to have ample justification. However, he has never shown evidence of further trouble, the choked disc has disappeared without leaving much optic atrophy and most of the protrusion of the brain has disappeared so that possibly the entire intracranial disturbance was due to edema of the brain from contiguous inflammation.

A cerebral hernia of the size present in this case without there having been an incision or injury to the dura does not occur. Very moderate sized ones appear when a decompression has been performed, without incision of the dura, for intracranial tumor. The only explanations that offer themselves are that the dura may have been accidentally cut in laying open the sinus or that the dura was necrotic and later broke down, throwing off a portion of the dura and creating a defect through which the edematous brain protruded. Nowhere in the literature reviewed has a similar hernia appeared except in cases where abscesses were searched for or drained, in which cases fair sized incisions were made in the dura. Another possible explanation of the appearance of the hernia is that there existed a subdural as well as an epidural (perisinus) abscess communicating by

a minute sinus. This would explain the rise of temperature for three days, occurring in the fourth day after the original operation, which temperature disappeared on the release of some pent up pus, possibly subdural in origin.

In considering the point of origin of the cerebrospinal fluid we are again confronted with a difficult problem. Had the point of leakage been found at the edge of the hernia, the explanation would have been comparatively simple, for it would have been most likely that the subarachnoid space would have been entered where it surrounds the neck of the hernia. However, this was not the case. It occurred at the summit of the hernia after a couple of thin slices that had been hardened had been removed so that there could not have been any contact with the dura or arachnoid. Two possible solutions offer themselves. The one is that a sulcus had been cut across that had been closed by adhesions above but had a channel beneath which communicated with the subarachnoid space. The other very possible solution is that the descending horn of the lateral ventricle, which may have been forced outward with the hernia, the brain tissue being thinned out over it, was accidentally entered when removing the slice of brain. This condition was found to explain the cerebrospinal fluid leak occurring in a case reported by Auvray, in which an unsuccessful search for a brain tumor in the occipital region was followed by a cerebral hernia from which there occurred a spontaneous discharge of cerebrospinal fluid. Subsequent autopsy showed prolapse of the lateral ventricle into the hernia as the source of the fluid.

The problem of treatment of the postoperative hernias is one that is far from settled. It appears that in many of the cases reported the pathology of the cause of the hernia is not taken into consideration. The operators appeared to presume that all that is necessary is to get rid of the mass protruding and no further trouble would be experienced. This, however, proved almost invariably not to be the case, for the hernia, in the great majority of the cases, recurred very promptly and attained the same or larger size than previously. These operators apparently lost sight of the fact that the hernia was caused by the cerebritis or cerebral edema present as the result of the infection or the traumatism caused by the operation. As a result the more operative work they did, the more

avenues for reinfection they opened, and the longer it would take for the hernia to subside. The remarkable part about the whole matter is that no fatal meningitis occurred as a result of cutting the hernia flush with the surface, especially since it was accompanied in quite a number of the cases by an escape of cerebrospinal fluid, showing that the subarachnoid space had been entered. Acquired immunity must play an important rôle in these cases of long standing infection.

The greater bulk of the operators appear to favor the waiting policy with success. The hernia appears to recede if kept clean and enough time allowed for the brain to recover from the infection. In those cases which did not recede within a reasonable time, a second brain abscess was usually found to be the cause. The most successful of the operators that resected the hernia were those that waited for quite some time and then tried immediate closure of the wound after doing the resection. When this was done early it was usually unsuccessful.

Among the operators advocating the less radical policy, various methods are used to assist nature in hastening the recession of the hernia. Stenger advocated spinal puncture to reduce the intracranial pressure. Milligan recommended spinal puncture, at the same time applying pressure over the hernia by a sterile copper or lead sheet. As an alternative method he advocated enlarging the bone wound around the hernia so as to release some pressure and allow the hernia to recede. Fagge, in discussing Hugh Jones' case, made a very pertinent statement when he said that he thought the hernia was not due to the removal of too much or too little bone but caused by a local and spreading infection, either due to another abscess or diffuse infection of the cerebral tissues. He at the same time threw out the suggestion, in 1902, that all of the area of brain superficial to an abscess should be removed, making the bottom of the abscess the bottom of the wound, thus making a funnel shaped excision of brain tissue. This has been elaborated, apparently entirely independent of this suggestion, by King in 1924, who has given the clearest, most minute and apparently the most successful method of handling the whole problem of brain abscess and herniation, to be taken up later. Van Hord used a silver plate to make pressure on the hernia.

Anastassiades rather indefinitely described but did not illus-

trate a contrivance made of a sheet of lead  $\frac{1}{2}$  mm. thick, the center of which was pressed over a hard cylindrical object the approximate diameter of the wound. This created a depression the thickness of the skull and wound in the center of the lead plate. This center part was then perforated in numerous places to allow for drainage from the wound. The outer part of the plate was made to conform to the shape of the skull and strapped in place. Thus, when the plate was placed with the center portion inverted in the wound, the discharges could gain access to the dressings but the brain could not protrude. In the case of deep abscesses he placed a glass drainage tube through the center of the plate to the bottom of the abscess. As the area of the wound closed in he had smaller plates to fit. He reported several successes with it.

One of the simplest methods of treatment reported is by Behrend, who stated that a few treatments of ten minutes' duration, at 20 cm. distance, with the caloric hot air douch fan (Electrischen Heissluftdusche Fön) tends toward early drying up and shrinkage of the hernia without the least inconvenience to the patient. Derganc advocated the application of X-ray in conjunction with spinal puncture.

As stated previously, the method which, in the opinion of the writer, is the most logical and most likely to give favorable results is that described by King in conjunction with his operation for abscess of the brain. In his operation he unroofs the abscess, allowing the brain to herniate and then treats the hernia. The entire treatment consists in keeping the hernia protected from injury and pressure in the early stages while keeping it bathed in Dakin's fluid. This is done through the fenestrations in a sheet of rubber dam which is placed in immediate contact with the hernia so as to cause no traumatism during dressings. Everything is done that will enable the edema and inflammation of the brain to subside. The sloughs that appear on the surface of the hernia are not forcibly removed nor cut off but allowed to separate spontaneously. When after a time the hernia becomes covered with clean granulations and is beginning to recede, he assists nature by strapping it with perforated adhesive plaster over which Dakin's dressing is applied. These should be changed daily. The entire scalp and ears must be protected by vaselin gauze. After the

first two weeks from the time of operation he uses judicious lumbar puncture at intervals to prevent excessive size of the hernia and rupture of the lateral ventricle. In the absence of any further infection, the hernia will recede and dermatize.

A year or more later, avoiding every chance of a lingering latent infection being still present in the wound, a scalp plastic is to be performed to eliminate the thin, poorly nourished scar tissue covering the hernia, to make proper preparation for a later cranioplasty, for protection and appearance. At the end of 1½ to 2 years a cranioplasty is performed.

The most serious objection to this method of treatment is the prolonged hospitalization and the excessive amount of careful nursing attention these patients require.

With reference to our own case, unless the hernia recedes from the external auditory canal allowing it to become covered with epithelium and dry, we fear to make any further effort to cover the defect with bone or any firmer tissue lest we start up a reinfection and possibly meningitis, etc.

In conclusion, the writer wished to express his gratitude to all who worked so faithfully with him in bringing this case to a successful issue, with especial mention of Drs. Fay, Yaskin, Smukler and Ginsburg.

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SEPTIC THROMBOSIS OF THE CAVERNOUS  
SINUS: REPORT OF TWO CASES.\*BY LYLE S. POWELL, M. D.,  
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Reports of cases of septic thrombosis of the cavernous sinus are still rare enough to warrant their publication in current literature, especially those cases in which autopsies have been obtained. Dorland Smith,<sup>1</sup> in 1918, after a careful study, found 140 reported cases. Of these, 56 (40 per cent) were secondary to ear infections; 40 (35 per cent) were secondary to face or orbital infections; 18 (13 per cent) were secondary to mouth and throat infections; and 13 (9 per cent) were secondary to nasal infection. Seven per cent were reported as having recovered. Langworthy,<sup>2</sup> Chisholm and Watkins,<sup>3</sup> Edward Jackson,<sup>4</sup> H. P. Mosher,<sup>5</sup> John Phillips,<sup>6</sup> John Myers,<sup>7</sup> Ewing Day<sup>8</sup> and others have made more or less recent and noteworthy contributions to the literature on this rare and dreadful malady. It is universally recognized that septic thrombosis of the cavernous sinus almost always follows infections of the face, nasopharynx, paranasal sinuses, teeth and middle ear.

Without going too deeply into the anatomic details, it may be said that the cavernous sinuses are paired venous sinuses lying on either side of the sella turcica on the body of the sphenoid bone directly under the middle portion of the cerebrum and almost directly posterior to the inner canthus of each eye. They connect posteriorly with the superior and inferior petrosal sinuses, and anteriorly they receive the valveless superior and inferior ophthalmic veins which in turn anastomose freely with the angular, frontal, supraorbital and facial veins, also valveless. Branches are also received directly or indirectly from the cerebrum, pterygoid plexus, ethmoid veins, sphenopalatine vein, pharyngeal plexus, posterior auricular

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occipital veins, vertebral sinuses and basilar plexuses. The following outline by Myers<sup>7</sup> is worth repeating, as it gives a comprehensive idea of the relation of these veins and the areas they drain to the cavernous sinuses:

#### THROMBOSIS OF THE CAVERNOUS SINUS.

| FROM                         | THROUGH  | OPENING IN SKULL                           |
|------------------------------|--|--|
| 1. Anterior Scalp            | 1. Frontal, Supraorbital, Facial, Angular and Ophthalmic Veins               | 1. Sphenoidal Fissure                      |
| Face                         |  |  |
| Neck                         |  |  |
| 2. Orbit                     | 2. Pterygoid Plexus  | 2. Foramen Ovale                           |
|                              | Ophthalmic Veins   | Sphenoidal Fissure                         |
| 3. Nose                      | 1. Ethmoidal Veins   | 1. Openings in Cribriform Plate            |
|                              | 2. Sphenopalatine Vein thru Pterygoid Plexus                                 | 2. Foramen Ovale                           |
|                              | 3. Anterior Facial, Angular and Ophthalmic Veins                             | 3. Sphenoidal Fissure                      |
| 4. Teeth                     | Pterygoid Plexus   | Foramen Ovale                              |
| 5. Tonsils                   | Pharyngeal Plexus and Pharyngeal Pterygoid Plexus                            | 1. Carotid Canal<br>2. Foramen Ovale       |
| 6. Neck and Occipital Region | Posterior Auricular, Occipital Veins, Vertebral Sinuses and Basilar Plexuses | 1. Condylloid Foramen<br>2. Foramen Magnum |

Anteriorly and posteriorly—that is, in front of and behind the sella turcica are the communicating sinuses connecting the right and left cavernous sinuses. Thus it is readily seen that there is formed about the upper aspect of the body of the sphenoid bone a circular sinus. The cavernous sinuses themselves, right and left, are trebeculated or honeycombed by numerous thin membranous splits from the dural walls. Lying within each sinus is the internal carotid artery, the sixth cranial nerve, and, along the lateral aspect, imbedded in the dural walls, lies the third and fourth cranial nerves and the ophthalmic and superior maxillary divisions of the fifth nerve. The sphenoid air sinus lies directly under the cavernous sinus on either side and is separated from it by the thin, bony wall of the sphenoid and the dura.

It may be readily appreciated that when a thrombosis or obstruction in the cavernous sinus has progressed to the point

where return circulation is interfered with to the extent that edema of the eyelids, chemosis of the bulbar conjunctiva and fixation of the eyeball has occurred, that the thrombus did not occur coincidentally with these symptoms. Certainly it has been in the process of formation for a greater or less length of time previously and that at the time when symptoms such as just described begin to appear the last free channel for the return blood flow has become obstructed. In other words, this thrombus is probably not quickly formed but one that forms progressively and by the nature of the sinus, trebeculated and honeycombed as it is, may have consumed a considerable time before actual obstructive symptoms appear. This may be the reason for the notable uneasiness and consciousness of impending trouble experienced early by the patient. Both the cases reported herewith had a feeling that all was not well, a premonition of impending disaster without any very notable or diagnostic symptoms. It also may be readily understood that, during the formation of this septic thrombus, involvement of the third, fourth, fifth or sixth nerve may occur early, depending entirely upon the whim of this particular infection and its localization within the sinus. It seems logical to believe, however, that the fourth nerve, lying free in the sinus, would be the most likely to be the first involved. This occurred in both these cases. As the infective thrombus progressed through the communicating sinuses to the opposite side, the first involvement of the opposite eye was the fourth nerve with paralysis of the external rectus. It has been stated repeatedly that the third nerve exhibits the earliest and most complete involvement. In both these cases ocular excursions were attempted in all but the outer quadrant until complete fixation of the eye by edematous proptosis. In neither of the two cases was there any neuralgic pain. In neither was there any interference with the muscles of mastication. In neither case were the ophthalmoscopic findings in the fundi at all commensurate with the external symptoms. Both cases exhibited albuminuria early, and in both cases there was an early or coincident septimecia.

Case 1.—Dr. W. B. G., age 38, had for several days a small furuncle on the internal lateral aspect of the vestibule of the right nostril. There was considerable redness and soreness

which progressed upwards. On the fourth day, August 24, 1926, the redness was somewhat increased and there was some tenderness as high as the bridge of the nose. He, however, regarded the lesion as inconsequential. On the afternoon of this day he was stricken with severe pain in the left kidney region, for which he consulted a physician, who sent him home to bed, applied heat to the region, forced fluids and gave aspirin as necessary. That evening he complained of a pleurisy like pain on inspiration. No chest findings could be ascertained. However, this chest pain became more severe, and the right side was strapped but was removed a few hours later. Pain during the night was so severe in both the right chest and flank that the patient took one-half grain of morphin by mouth and repeated in one and one-half hours. Next morning the pain was quite as severe as the day before. The physician, thinking of a Deitl's crisis, gave him an enema in the knee-chest position with good results, which seemed to alleviate the pain somewhat. That night the patient suffered some pain in the chest and kidney region but not so severe as before. The following morning, August 26th, the third day after having consulted a physician, and the seventh day following the appearance of the furuncle in the nose, the author was called in consultation on account of edema of the right eyelids which had gotten progressively worse since early morning.

On examination, the right eye was found to be fixed, there was a considerable degree of exophthalmus, there was marked edema of the upper and lower lids and cheek region down as far as the angle of the jaw. The bulbar conjunctiva was markedly chemotic. The pupil was equal in size with its fellow, was regular and reacted to light and accommodation. The consensual reflex was present, right and left. There was no anesthesia of the cornea. There was very little if any engorgement of the retinal veins and no edema of the disc. Ewing's sign was negative. There was considerable redness and tenderness extending upward along the right side of the nose, as high as the inner canthus. The fununcle inside the vestibule of the nose had ruptured and a small bead of serum was present on its summit. There was no other intranasal pathology. The paranasal sinuses were negative. Teeth, mouth, throat, scalp and neck were negative. The left eye was entirely normal in

its excursions, reflexes, appearance and function. The patient was obviously septic. There was a fine maculopapular rash on the chest. Pulse was 110, bounding in character. Temperature was 102, blood count—reds 4,800,000, whites 10,000, 90 per cent polys. A diagnosis of cavernous sinus thrombosis was made. A spinal puncture and blood culture was recommended but refused by the patient. He was put on urotropin, forced fluids and morphin as necessary.

By evening of the same day the exophthalmus of the right eye had increased, the bulbar conjunctiva was prolapsed irretrievably between the palpebral margins, the patient began to complain of severe headache. At this time there appeared a total paralysis of the external rectus of the left eye. All other ocular excursions appeared normal in the left eye. The patient had by this time diagnosed his own malady and insisted on conference with various persons concerning his private affairs, as he foresaw fatal termination.

Next morning, Friday, there was slight edema of the upper lid of the left eye, which increased progressively during the day. By night there was equal edema and exophthalmus of both eyes and cheek region with total fixation of both globes. Due presumably to the administration of narcotics for relief of the headache, the pupils were pinpoint and the fundus could not be observed. The pupils, however, still reacted to light and accommodation, the consensual reflexes were present, there was total anesthesia of the right cornea, partial of the left. By this time no response was noted on either upper lid when the patient attempted to elevate them. Atropin was instilled into both eyes in order that the fundi might be observed. The fundi of both eyes appeared surprisingly normal. There was slight engorgement of the retinal veins of the right eye, with slight edema of the retina and none of the disc. The fundus of the left eye appeared entirely normal. Temperature was typically septic, picket fence type, with an upper limit of 103.6. The pulse ranged between 100 and 130. The patient was entirely conscious until this time, although he complained bitterly of headache. Friday night he became irrational at times and exhibited mutterings, twitchings and picking movements of the hands, typical of meningeal involvement. The patellar and

knee jerk reflexes were absent and Babinski's sign positive on both extremities.

Saturday the patient was in a state of semicomatose continually. The exophthalmos in both eyes remained unchanged, the bulbar conjunctiva was prolapsed between the palpebral margins in both eyes, the entire face and neck down to the level of the cricoid cartilage was markedly swollen and edematous. Although the cornea of both eyes had been anointed frequently with sterile vaselin, the right cornea was very hazy, and the fundus could not be seen distinctly. The left cornea, however, **remained clear**. There was no edema or choking of the disc, **there was very little, if any, engorgement of the retinal veins** and no edema of the retina could be detected. There were no hemorrhages in the retina. It was a strikingly normal fundus under the circumstances.

Sunday the condition of the patient remained unchanged. The fundus of the left eye presented the same picture as before. Consent for spinal puncture was obtained from the family at this time, which was accordingly done. The fluid was under 300 mm. (water) pressure and markedly purulent. There were 2,500 cells, globulin positive. Pneumococcus was found both on culture and direct stain.\* Sunday evening at 6 o'clock the pulse dropped to 80, at 8 o'clock to 60, and respirations were labored. The patient died at 7 o'clock, Monday morning. Autopsy was not permitted.

Thursday the patient was given 35 cc. mercurochrome, 1 per cent, intravenously; Friday, aolan, 10 cc., intramuscularly; beginning Friday night, 10 cc. leukocyte extract intramuscularly every eight hours for three doses.

It is a noteworthy fact that this patient, being a hay fever victim, had for the previous two weeks inoculated himself with various and sundry hypodermic remedies for this condition in a desperate effort to alleviate his nasal condition. This may have reduced his general resistance somewhat. It is also noteworthy that this patient had had a premonition of death for two weeks preceding his demise and had discussed and outlined financial matters with his family with all the certainty of a doomed man.

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\*Laboratory work done by Dr. J. L. Lattimore, Topeka, Kans.

Case 2.—Miss E. J., age 18, white, attended a parade of university students on the night of October 8, 1927, at which time she contracted a severe "cold," or, rather, as she stated, "caught more cold," saying that she had had a slight "cold" in the head with nasal discharge for a week previous. Following this second "cold," she felt unusually bad. She had some pain over the right side of her face and eye. At this time she consulted a physician. There was no redness or swelling, but the girl stated that from the way the eye felt she must have gotten something in it, but nothing could be found. A colloidal silver solution was prescribed to be instilled into the conjunctival sac. During the next few days she was more or less incapacitated and confined to her home by her "cold" and a deep seated pain behind the right eye. On October 17th, while at her mother's home in an adjoining town, she consulted another physician, who found her with slightly increased temperature, slight headache and general symptoms of what is commonly called a cold. On October 18th, symptoms remained the same except that there was slight redness of the right eye and the W. B. C. 32,000 (polys 90 per cent) and temperature 104. The next day she returned to Lawrence and consulted another physician. At this time there was slight edema of the right eyelid, the bulbar conjunctiva was injected and reddened, the patient complained of considerable pain deep in the right orbit. The patient was hospitalized and the author called in consultation.

On examination, the right eye was found to be considerably proptosed. There was some edema and considerable redness of the bulbar conjunctiva. The pupil reacted to light and accommodation. The cornea and the media were clear. There was no edema or papillitis of the disc; there was no edema of the retina, but a slight engorgement of the retinal veins was detected. Very limited excursions of the eye were possible in all directions except externally. These movements seemed to be limited by the edema and proptosis. There was a total paralysis of the external rectus. Diplopia was experienced except when looking straight ahead. Patient said she had seen double continuously for past twenty-four hours and occasionally for the past three or four days.

The left eye exhibited a total paralysis of the external rectus; other ocular excursions were normal; pupil was equal with its fellow and round and regular, and reacted to light and accommodation. The consensual reflexes were present, right and left; there was no redness or injection of the bulbar conjunctiva; the cornea, media and fundi were entirely normal. There was no anesthesia of either cornea. The nasal septum was badly deflected to the right. In the right middle meatus was a string of thick, creamy pus which reappeared on wiping away and which on culture proved to be a staphylococcus aureus. The mucous membrane of the septum and turbinates of the right nostril was reddened and thickened. There was no pus in the left nostril and the mucous membrane appeared normal. On transillumination, the right frontal and maxillary sinuses were totally dark, while those on the left transilluminated perfectly. X-ray examination showed a marked clouding of the right antrum, ethmoids, with questionable involvement of the sphenoid and right frontal. The paranasal sinuses on the left were entirely clear. Neither the right nor the left auricle, external canal or membrana tympani exhibited any signs of pathology. There was, however, marked edema of the right facial region down as far as the angle of the jaw. There were a number of herpetic lesions on and about the lips. The teeth were exceptionally well kept, but there were several small ulcerated patches on the gingival margins from which bright red blood was oozing. The soft palate was in the midline; the excursions of the tongue were normal in every direction. The patient blew out either cheek with equal facility; there was no drooping or facial asymmetry. The back of the neck was rather stiff, although not rigid, but the patient complained when the chin was brought down onto the chest. Kernig's sign was positive; knee jerks were present, perhaps a trifle exaggerated. Abdominal reflexes were present, right and left. The pulse was 112, temperature 105.2, respirations 24. The patient responded readily to questions and was very much concerned as to her condition, but at the same time seemed more or less stuporous. A diagnosis of cavernous sinus thrombosis with meningitis was made. Ephedrin inhalant was sprayed into the nose every three hours, a saline mouth wash, forced fluids, aspirin, grains 10, p. r. n., and vaselin to the lips



and boric acid ointment to eyes were prescribed. A catheterized specimen of urine was ordered, which showed four plus albumen. Blood count 32,000, 90 per cent polys. Temperature was septic, with an upper limit of 105°. The spinal fluid was under greatly increased pressure, was cloudy and 850 cells were found by direct count. Patient was put on hexamethylamin, gr. V. q 3 h and morphin, gr.  $\frac{3}{4}$  p. r. n. The next morning there was some edema of the left upper eyelid, some chemosis of the bulbar conjunctiva and some exophthalmus. Both pupils, however, remained active to light and accommodation. There was no anesthesia of either cornea. The pupils were equal, round, regular, of normal size. Both cornea and media were clear; the right fundus showed moderate engorgement of the retinal veins; the left fundus appeared entirely normal. There was no involvement of either disc. There was marked edema of the right side of the face and neck down as low as the cricoid cartilage and on the left to the angle of the jaw. The neck was markedly stiffened, the patient's body could be lifted from the bed with the hands under the occiput. There was considerable drainage of thick, yellow, stringy pus from the right nostril. A considerable amount of the same purulent material was constantly seen in the posterior nasopharynx. The patient was muttering and semidelirious. The knee jerks were greatly exaggerated. Babinski was negative. The temperature was septic and ranged between 102 and 104 axillary. The pulse was rapid, 110 to 120, thin and of a dicrotic nature. During the afternoon of this day the patient complained of severe headache, which was relieved by morphin.

The next morning, October 21, 1927, there was complete fixation of the left eye; the bulbar conjunctiva was irretrievably prolapsed; there was enormous edema and proptosis of both eyes. There was, however, no real ptosis, even under this great tension. The patient was able to raise the upper lids somewhat when requested. A second spinal puncture was done, which revealed the fluid to be under greatly increased pressure and quite cloudy. The cell count was 1,200. Blood culture taken at this time was reported positive for staphylococcus aureus. Twenty cc. of 1 per cent aqueous solution of mercurochrome was given intravenously at this time with very little reaction.

The next day the condition remained unchanged except that the facial edema seemed to subside somewhat, presumably due to the establishment of collateral circulation. The patient complained of griping pains in the abdomen and began to have involuntary bloody stools. Although the boric acid ointment had been consistently applied to both cornea from the first, the right cornea was now quite hazy and the fundus was seen with difficulty. There was a crescentic defect in the cornea in the lower quadrant along the limbus. The left cornea remained clear. The fundus appeared normal except for a slight engorgement of the retinal veins. The arteries seemed more thready than the day previous.

October 22nd the condition remained unchanged, the patient being in a semidelirious condition, responding more or less coherently to questions. The pupils still reacted to light and accommodation. There was slight anesthesia of the right cornea; none could be detected of the left. The pupils were pinpoint, due presumably to the repeated administration of morphin. Atropin was instilled in both eyes the better to view the fundi. Involuntary passages of urine and feces continued, but no more blood was detected in the feces.

October 23rd, general condition unchanged except for the mental state, which was growing rapidly worse. Intelligent response to questioning was only occasional. Paregoric was administered every three hours in an effort to splint the bowel and check the frequent involuntary defecations.

October 24th and 25th, the condition was unchanged.

October 26th, patient was alternately stuporous and delirious. The right fundus could be seen very indistinctly. The left cornea and media were clear and the fundus was distinctly seen. There was slight edema but no hemorrhage of the retina. The retinal veins were moderately engorged, the disc appeared normal. There was total anesthesia of the right cornea, partial of the left. The elbow and knee reflexes were entirely absent, Babinski positive; there was marked ankle clonus, right and left. The pulse dropped to 88, but in the evening went up again to 128; the temperature ranged between 94 and 101.6 axillary. Respirations were shallow and rapid. At 12:30 midnight the temperature was 101.6 axillary, pulse 100, respirations 18. The patient expired at 2:35 a. m., the eighteenth day after first

consulting a physician for her "cold" and pain in the eye, which was undoubtedly due to a right pansinusitis, and the tenth day of hospitalization. During the entire illness the urine showed four plus albumen.

Autopsy.—On removing the skull cap, the dural vessels were noted to be engorged. An acute diffuse purulent leptomeningitis was present. There was some flattening of the convolutions. The cortex was quite soft, almost mushy to the touch, but solid enough to retain its form when handled. In the right temporoparietal region there was a large subdural abscess approximately 2 cm. in diameter involving the cortex. A similar abscess was found in the left temporoparietal region, a little higher and a little nearer the vertex than that on the right. The pus in both cases was very thick, stringy, yellowish, of about the consistency of corn syrup. The frontal lobes were lifted and found to be floating in a veritable lake of the same sort of pus. The cerebrum was removed, the cavernous sinus opened carefully and found to be entirely occluded by old thrombi of brownish yellow color. The thrombi extended on the right through the superior and inferior petrosal sinuses and back into the lateral sinus as far as the knee. On the left the thrombus extended but 2 or 3 mm. into the inferior and superior petrosal sinuses. There was no thrombus in the left lateral or sigmoid sinus. The ventricles did not appear unduly distended and contained clear, straw colored fluid. The choroid plexuses seemed markedly enlarged. The tentorium was removed and the cerebellum was found to be considerably more softened than the cerebrum and was entirely surrounded by the thick, yellowish pus described above. The brain stem was severed as low as possible in the foramen magna and entire cranial contents prepared for histologic examination.\* Cultures and smears of the pus showed staphylococcus aureus.

The right frontal sinus was opened from above and found to contain thick, almost jellylike, yellowish purulent material, as did also the right sphenoid, the right ethmoids and the right antrum. The left frontal, ethmoids and antrum were entirely clear. The left sphenoid was filled with the same thick, almost

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\*Laboratory work done by Drs. Sherwood and Downs, Department of Bacteriology, University of Kansas College of Medicine.

jellylike pus as the right sphenoid. The chest and abdomen were not opened.

#### MICROSCOPIC REPORT.

Meninges hyperemic and thickened, covered with thick purulent exudate. Thrombosis of meningeal vessels. One shows a partly organized septic thrombus. Two other vessels show partly organized thrombi not septic.

Cerebral cortex: Several areas of hemorrhage, rather superficial. Areas of noticeable vacuolization. Many nerve cells show degenerative changes or are absent. The polymorphonuclear leucocytes not distributed throughout the cortex. The acute inflammatory reaction seems to be confined to the meninges.

Cerebellum: Degeneration evidenced in certain areas by marked vacuolization, formation of granular debris and alteration in staining reaction.

Bacteriologic results: Blood culture, staphylococcus aureus; pus culture, staphylococcus aureus; spinal fluid, negative on culture.

Septic thrombosis of the cavernous sinus has been classified (Eagleton<sup>9</sup>) according to the invasion as either anterior or posterior, those in the latter class coming almost always following aural infection with a subsequent thrombosis or periphlebitis of the lateral sinus with the extension through the petrosals into the cavernous. Obviously the two cases just reported were anterior invasions, the first case arising from a small furuncle in the nose with an ascending periphlebitis. Most likely there was an actual thrombosis of the ophthalmic veins and possibly of the angular, which extended directly into the cavernous sinus. This first case may be correctly called fulminating in type, offering little or no reasonable hope from surgical procedure. The prodromal chest and flank pain was undoubtedly due to septic infarcts.

In the second case, while no actual route of invasion could be demonstrated at autopsy, most probably infection occurred by extension through the ethmoid veins. It is possible, however, that there was a direct extension by continuity of tissue from the sphenoid air sinus directly into the cavernous sinus, although no dehiscence of the upper sphenoid wall could be demonstrated. At the time this patient was first seen a septic

meningitis had already developed, and surgical procedure, even on the paranasal sinuses, was not considered expedient. It is possible, however, that if this patient had been seen early and the paranasal sinus disease diagnosed and surgical drainage instituted, the sinus thrombosis would not have occurred.

The impression gained by the author from a thorough review of the literature on the subject is that surgical procedure directed at drainage of the cavernous sinus is hardly justifiable, especially in view of the difficulty of approach and the multilocular nature of the sinus. In very expert hands and in favorable cases seen early without meningitis, surgery may, however, prove beneficial. An early diagnosis of septic thrombosis of the cavernous sinus, once made, the obvious duty of the attending physician is to permit the patient to settle, insofar as possible, his worldly affairs before the onset of coma.

Attention is invited to the following points:

1. Potential danger of eye, nose, face and ear lesions.
2. Importance of eye findings in establishing early diagnosis, especially derangement of the extrinsic ocular muscles.
3. Astonishingly slight dérangement of the retinal circulation.
4. Early premonitory symptoms, possibly due to the nature of the periphlebitis or thrombus and the coincidental septicemia.
5. Futility of surgical intervention because: First, of the coincidental pathology (meningitis, septicemia, etc.); secondly, the difficulty of certain approach to the sinus; and thirdly, the multilocular nature of the sinus itself.

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LXXVI.

RHINOGENIC FRONTAL LOBE ABSCESS: REPORT  
OF TWO CASES.\*

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Frontal lobe abscess complicating nasal accessory sinus sup-  
puration is rather infrequent. There are less than one hundred  
and fifty cases reported in the literature, yet it is the com-  
monest intracranial complication of suppurative frontal sinus-  
itis.<sup>1</sup> The disease is usually characterized by the absence of  
symptoms, the diagnosis often being suggested by a persistent  
headache following frontoethmoidal surgery. Since 1925 three  
new operative procedures have been described by King, Els-  
berg and Cahill that should replace the older methods of  
drainage, and should materially reduce the mortality.

Case 1.—G. E., a farmer, age forty, was referred by Dr.  
E. M. Welty, April 8, 1925. The patient states that four  
months previously he had developed a "head cold" with pain  
and tenderness over the right eye which has persisted. At  
that time two of his children had scarlet fever. The patient,  
himself, had a sore throat and temperature, but no rash. He  
was treated by his local physician for a profuse purulent dis-  
charge from the right nostril, and a swollen and painful eye.  
Early in February the symptoms abated somewhat, and later  
became more aggravated. Two months ago he began vomit-  
ing unrelated to meals, though usually before breakfast. The  
frontal headaches increased in severity, radiating backward  
toward the occiput on the right side, and always were worse  
just before he vomited. Impairment of vision and vertigo  
appeared about the same time, and for the past two weeks  
has had some fever. Since the onset of the disease, patient  
has complained of an annoying backache.

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\*Presented as a candidate's thesis to the American Laryngological,  
Rhinological and Otological Society.

There was marked tenderness on pressure over the anterior wall and floor of the right frontal sinus. Edema was absent. Thick greenish pus was seen pouring out from the right middle meatus. The vision of the right eye was 20/40 and of the left 20/30. The fundus revealed a bilateral papilledema measuring three-quarters of a millimeter of swelling. Multiple hemorrhages were present in the right eye. A sector shaped scotoma was found in the temporal field of vision in the left eye extending to the midline. No attempt was made to determine his turning or caloric reactions because of his generally weakened condition. The neurologic examination was negative.

He was sent to the hospital with a diagnosis of chronic purulent frontal sinusitis, and a probable frontal lobe abscess. On admission, his temperature was 98.6; pulse 74; respirations 22, and within twenty-four hours his temperature rose to 101.4; pulse 56; respirations 18. The pulse rate continued to range between fifty-six and seventy until after his operation. The white cell count was 16,000. The spinal fluid was clear, measured 30 mm. of pressure, and contained five cells to the field. The blood and spinal Wassermann tests were negative.

The patient was operated upon by Dr. E. M. Welty and the writer, April 10. An incision was made through the right brow, flaps retracted, and frontal sinus entered. The sinus was filled with thick greenish pus under pressure, and the mucosa found greatly thickened with polypoid changes throughout. A large spicule of necrotic posterior plate was found free in the sinus. Through a perforation in the posterior plate about the size of a dime, the dura could be seen covered with necrotic granulations, and pus was exuding from the center of this area. The fistula through which the pus came would not admit a brain needle. This was enlarged, and three ounces of thick greenish pus evacuated from the frontal lobe. A pure culture of streptococcus hemolyticus was obtained. The needle under its own weight was allowed to enter a distance of three inches, but did not reach the bottom of the cavity. Rubber tubing was inserted for drainage and sutured to the skin flap. The sinus was small and the supraorbital ridge and floor were removed, the anterior ethmoids were broken down, and a large opening made into the nose. Iodoform

gauze packing was used, and carried into the nose. The lower angle of the wound was closed.

The wound was dressed daily and, except for one rise in temperature, the convalescence was uneventful. The drainage tube was gradually shortened and on the twenty-sixth of May removed entirely. The sinus closed quickly, and on the fourth of June the wound was entirely healed. The patient was discharged on the seventh in good condition.

December 21, 1925, patient gave normal responses to turning tests. The fundi were negative, but the fields showed a left lateral hemianopsia. Vision, O U, 20/70, which was corrected to 20/20 O U with lenses.

Patient was last seen September 12, 1927, when there had been no recurrence of symptoms.

Case 2.—E. B., male, age 14, was referred by Dr. E. J. Barnett, August 8, 1925, complaining for the past three days of pain in the nose and right orbit; swelling and tenderness of

Examination by Dr. R. J. Sprowl showed marked swelling and edema of the right upper lid and extreme tenderness over the right frontal and ethmoid areas. There was edema of the bulbar conjunctiva on the temporal side. The ocular the right upper lid.

movements were normal. Pupils were equal and reacted to light and accommodation. The fundi were negative. Vision, O U, 20/20. Pus was seen exuding from the right middle meatus and there was some yellow postnasal discharge. A diagnosis of orbital cellulitis complicating an acute ethmoiditis was made, and patient removed to the hospital.

On admission, temperature was 104.2, pulse 124, and respiration 26. The leucocyte count was 18,700. The urine showed a faint trace of albumin and a few pus cells. Roentgenologic report by Dr. Arthur Betts showed the right antrum and both ethmoid areas cloudy; frontal sinuses small.

There was a slight improvement the next day, but on the following morning the temperature reached 104, and operation was advised. Under general anesthesia, a brow incision was made and a large orbital abscess evacuated. The floor of the frontal sinus and the inner orbital plate were carefully inspected, but no area of necrosis was found. A cigarette drain was inserted, and the wound left open.



The patient improved steadily, but the swelling decreased slowly. The intraocular findings were normal. The nose was treated daily, discharged freely, and headache was absent. On the third of September the patient was discharged from the hospital, although there was a slight serous discharge from the wound. The nasal discharge had entirely ceased.

On October 13 the wound still discharged slightly and an X-ray picture was taken. The report read as follows: "There appears to be some erosion of the frontal bone above the sinus in the midline. There is an unusual shadow along the superior wall of the right orbit which might be due to bone destruction."

On October 20 the child appeared again complaining of parietal headache, and said he did not feel well. The father stated he had lost his appetite, and had vomited once. The sinus was probed, and roughened bone could be felt. Considerable tenderness existed over the frontal bone above the sinus, and there was some edema of the overlying skin. The temperature was 98; pulse 84. Neurologic examination was negative. Nystagmus on turning to the right lasted fourteen seconds, and on turning to the left it was present for eighteen seconds. The right disc showed a beginning optic neuritis with engorgement of the veins, and there was a slight temporal haziness of the left disc. V, O S 20/20, V, O D 20/20—2. The visual fields were normal. The nasal examination was negative. A diagnosis of osteomyelitis of the frontal bone with possible brain abscess was made, and the patient sent to the hospital for operation. Temperature on admission was normal; pulse 80; leucocyte count 16,400.

The following day the old incision was opened and enlarged and spicules of necrotic bone found under the skin. The outer angle of the small frontal sinus showed necrosis of both the inner and outer plates. There was a small perforation of the posterior plate, and the adjacent dura was covered with necrotic granulations. The mucosa of the sinus was thickened and necrotic but no pus was seen. The outer plate of the frontal bone and the cellular structure under it, reaching well above the sinus and toward the midline, showed an osteomyelitis. Upon enlarging the perforation the dura bulged forward into the wound, appeared to be quite tense, and the con-

volution indistinct. Pus had not been encountered at any time, but it was thought advisable to open the dura as the symptoms had not been accounted for. The wound was carefully sterilized, and an incision one-half inch long made in the dura. Three puncture wounds were made into the brain substance for a depth of two centimeters, but no pus found. The dural incision was left open and surrounded with iodoform packing. The opening into the nose was enlarged, and the anterior ethmoid cells were broken down. A gauze drain was carried into the nose and the external wound was left open.

The dressings were changed daily, and there was cerebrospinal fluid drainage for three days. On the third day there was a small hernia cerebri about the size of a pea. The neuritis in the right eye had increased to one-half millimeter of swelling with a small hemorrhage on the nasal side of the disc. On the sixth day the patient complained of some nausea and the hernia had increased slightly in size. Spinal fluid was clear, under twenty-eight millimeters of pressure, and contained twenty cells to the field. Dr. George Downs was called in consultation and advised delaying further operative procedure. Five days later the spinal fluid was under thirty millimeters of pressure, but remained clear. On the fifteenth day the patient complained of severe headache and nausea. The Babinski response was suggestive for the first time and the papilledema had increased to one and one-half millimeter of swelling.

The following morning, under general anesthesia, Dr. Downs and the writer reoperated upon the patient. The old incision was enlarged and a vertical incision carried upward over the frontal lobe. Considerable softened bone was removed, exposing a large area of dura. The dural wound was enlarged upward, and a brain searcher was introduced just above the hernia and an encapsulated abscess located one inch below the surface. The cavity was unroofed as described by King, and one-half ounce of pus evacuated. The wound was dressed daily, but not Dakinized as King suggested. The hernia was covered with rubber tissue coated with sterile vaseline and a gauze dressing applied over this. On the third day the hernia was quite large and spinal punctures were begun to reduce

the intracranial pressure, whereupon the hernia slowly reduced in size and the papilledema subsided. The general condition of the patient remained good, and on January 5 the wound was entirely healed. He was discharged from the hospital two days later.

He was kept under observation and remained free from symptoms until January 25, when he returned, complaining of severe pain in and around the eyes. He appeared ill and had vomited the previous day. The leucocyte count was 20,400, with 88 per cent polymorphonuclear cells. The discs were normal except for slight blurring at the upper pole in each eye, and fullness in the veins. Neurologic examination revealed a slight Kernig and diminished knee jerks. The wound was bulging slightly, and the brain seemed quite tense. The patient was returned to the hospital at once.

By the following day the Kernig had increased; the discs showed more blurring, and the pain in and around the eyes continued. Spinal fluid showed twenty millimeters of pressure, with 3,500 cells to the field, globulins + + +, and 66.5 milligrams of sugar.

On January 27 we again opened the old incision. Some inflammatory tissue was found under the scar, and the brain was bulging from increased intracranial pressure. A brain searcher directed backward located a large superficial abscess containing one and one-half ounces of thick green pus. A pure culture of pneumococcus was obtained. The abscess cavity was unroofed as in the previous operation, and the brain allowed to herniate.

The patient again had a prolonged convalescence which was otherwise uneventful except for a large hernia that was gradually reduced with spinal taps. On the nineteenth of March the wound was completely healed and he was discharged from the hospital on the twenty-sixth. When last seen, February 12, 1927, he was perfectly well.

Gerber<sup>2</sup> in 1909, after a careful review of the literature, collected 240 cases of intracranial complications following disease of the nasal accessory sinuses, 65 of which were frontal lobe abscesses.

Hajek<sup>3</sup> reports that Boenninghaus "proceeding rather critically in acknowledgment of the cases" was able to collect 359

cases of intracranial complications following sinus infections previous to 1923. Toti reported 530 in his collection. Hajek's statistics, taken from these reports, show 108 cases of cerebral abscess following frontal sinus suppuration, all, with one exception, being situated in the frontal lobe.

Eagleton's<sup>4</sup> collection of 140 frontal lobe abscesses contained those of traumatic, otitic and osteomyelitic origin as well as those due to frontoethmoid disease. Since 1921 cases have been reported by Friedman and Greenfield, Bellomo, Berry (ethmoid origin), Tarasido, Carpenter (two cases), Jessaman, Salinger, Parrish (two cases, one of ethmoid origin), Boss (two cases), Arnoldson and Bostrom, Cusatelli, Motley—a total of seventeen, including the two reported in this article. This would indicate that frontal lobe abscess is of infrequent occurrence, but as Leegaard pointed out, it arises more frequently than was supposed several years ago. No doubt quite a number have been operated upon and not reported, while many more have not been recognized.

In adjacent abscess the most frequent route of infection following frontal sinus suppuration is by direct extension with necrosis and perforation of the posterior plate. In Gerber's statistics, 44 of the 65 cases showed a perforation of the posterior plate. The next most frequent route occurs through a thrombophlebitis of the veins of the lining membrane of the sinus which anastomose with the dural veins. In this case there may be no macroscopic changes in the bone, and the adjacent dura and cortex appear normal. A complicating osteomyelitis may, through a thrombophlebitis of the diploic and dural veins, produce a brain abscess, and this type of abscess may be situated at a considerable distance from the original focus.

Leegaard<sup>5</sup> in his noteworthy monograph considered headache, vomiting, vertigo, slow pulse and optic neuritis the outstanding symptoms of frontal lobe abscess. He, however, emphasized Gerber's statement that the most characteristic feature of this disease was the absence of symptoms. When symptoms are present headache is the most constant, although its location is rarely of diagnostic value. Its presence, with increasing intensity, following intra- or extra-nasal surgery on

the sinuses should suggest intracranial involvement. Vomiting may occur early in the disease, and should always arouse suspicion, particularly if not related to the taking of food. Its importance cannot be regarded lightly when it follows sinus surgery. Slow pulse is an important sign, but is occasionally present in nasal accessory sinus disease without intracranial complications. As pointed out by Leegaard, if the patient has been under observation for some time, a decreasing pulse rate is significant. In our first case the pulse rate dropped as the temperature went up, and was considered extremely important. Vertigo is frequently cited in the literature, but less commonly so than vomiting. Optic neuritis and papilledema when present are positive signs. Eagleton found definite nerve head changes only three times in over one hundred reported cases of frontal lobe abscess. Hajek states that optic neuritis and choked disc have been frequently noted. Both of the cases reported in this article and five of those collected from the literature since 1921 showed either optic neuritis or papilledema. Psychic disturbances are not characteristic, but do occur; namely, apathy, irritability, and lethargy. In Harris<sup>10</sup> case apathy was a prominent symptom, but was erroneously ascribed to other causes. Eagleton regards convulsions as diagnostic, while Leegaard does not consider them of unusual importance.

A review of the literature impresses one with the distressing fact that the antemortem diagnosis of frontal lobe abscess was usually made during a radical operation on the frontal sinus, or shortly afterward and not clinically before operation, while an appallingly large number were diagnosed at autopsy. If an early diagnosis is to be made one must rely upon the etiology and symptoms of increased intracranial pressure. Morbid changes in the posterior wall of the sinus, and especially the adjacent dura, are strongly suggestive of intracranial involvement. In the absence of such changes, surgery should be delayed. If, however, the symptoms increase in severity, even in the absence of such morbid changes in the dura, exploration is indicated. A spinal puncture at this stage will frequently show increased pressure and a pathologic cytology. Pneumoventriculography as described by Dandy was diagnostic in Carpenter's two cases. In a large proportion of cases the

diagnosis will be in doubt until operative exploration. To quote Eagleton:<sup>7</sup> "As in a large number of the reported cases of frontal lobe abscess the patient died suddenly before a positive diagnosis had been made, operation must be performed while the diagnosis is still largely problematic, and consequently must be more or less exploratory."

In January, 1925, King<sup>8</sup> described a new operation for brain abscess and reported four consecutive recoveries. A large trephine opening is made over the suspected site, the abscess cavity located and the pus evacuated. The overlying cerebral tissue is excised, the abscess cavity being completely "unroofed" and the brain allowed to herniate. To prevent infection, the wound is irrigated frequently with surgical chlorinated soda solution. Excessive herniation and cerebral edema are controlled by repeated lumbar punctures. Epithelization is complete in from two to three months. Elsberg<sup>9</sup> the same year described his operation for brain abscess without limiting membrane. Having located the abscess, an incision three to five centimeters long is made in the cortex and the cavity thoroughly exposed. A piece of rubber dam is inserted into the cavity and gauze wicks are packed inside, thus forming a Mikulicz tampon, which holds the cavity widely open. More recently Cahill<sup>10</sup> reported twelve recovered cases of brain abscess drained with Mosher's meshed wire cone. The drain is inserted into the cavity and sutured to the dura and skin. Iodoform gauze is packed around the cone. Granulations soon hold it in place and as the cavity heals the cone is expelled. Motley reports two recoveries following this method.

In our first case a fistula was found leading from the abscess through the dura, back into the frontal sinus and required nothing more than sufficient enlargement of the fistula to admit a rubber drainage tube. In this type of case, surgery should be limited to simple drainage and the mortality should be very low.

The results following operations on the frontal lobe are not favorable, the mortality is high. According to the collections of Gerber and Boenninghaus, as reported by Hajek,<sup>11</sup> of the 108 cases of frontal lobe abscess following suppurative of the frontal sinus, 55 were operated upon and 20 recovered. Hajek

increased this number by the following reported cases: Bella, Protti, Broti, F., Fraser, S. S., Charles Imperatori, Linck, E., Corneil, P. Of the seventeen cases collected and reported by the writer, seven recovered. Eagleton<sup>12</sup> believes that the mortality remains about 80 per cent because of the difficulties of early diagnosis and our imperfect methods of treatment.

## SUMMARY.

1. Frontal lobe abscess, though rare, is not as infrequent as generally supposed.

2. The disease is frequently characterized by absence of symptoms.

3. The early diagnosis must be made on a history of nasal accessory sinus suppuration and symptoms of increased intracranial pressure.

4. Frontal lobe abscess with fistula requires only drainage, and recovery should be the rule.

5. Frontal lobe abscess, with or without limiting membrane, should be operated upon by one of the newer methods, as described by King, Elsberg or Cahill. The writer is greatly impressed with the one described by King.

6. Following these methods, the mortality should be materially reduced.

ABSTRACTS OF CASES OF RHINOGENIC FRONTAL LOBE ABSCESS  
COLLECTED FROM THE LITERATURE SINCE 1921.

Friedman, J., and Greenfield, S.: Frontal Lobe Abscess Secondary to Sinusitis. *Laryngoscope*, Vol. XXXII, 608, 1922. Child, age 11. Acute left frontal sinusitis. Exophthalmos. Radical left frontal operation. Pus and necrotic floor. Posterior plate appeared healthy. Temperature. Irrational. Chill. Left facial paralysis. Contracture left upper and lower limbs. Veins of fundus full. Complete left hemiplegia. Operation refused. Death. Autopsy. Right suppurative frontal. Inner plate normal. Dura thickened. Right frontal lobe abscess. Culture streptococcus hemolyticus. Left frontal lobe normal.

Bellomo, E.: A Case of Abscess of the Frontal Lobe From Fronto-Ethmoid Sinusitis. *Osp. maggiori*, 10, 131-136, 1922. Woman, age 19. Acute right frontoethmoidal sinusitis. Exophthalmos. Limited ocular movements. Ethmoidectomy. Fol-

lowed by headache, temperature, cyanosis and death. Autopsy. Right frontal and ethmoids contained pus. Dura adherent to right frontal lobe. Cortex soft. Abscess size of a nut in right frontal lobe.

Berry, Gordon: Brain Abscess of Paranasal Sinus Origin. *Laryngoscope*, Vol. XXXIV, 346, 1924. Woman, age 22. Acute left frontoethmoidal sinusitis. Five weeks later, convulsion, headache, vomiting, stupor and slow pulse. Left optic neuritis. Paralysis left external rectus. Endonasal drainage of left frontal, ethmoids and sphenoid. Punch removal of granulation tissue. Three weeks later, opened a brain abscess into nose located over left ethmoid roof. X-ray showed abscess cavity filled with air and located in left frontal lobe. Symptom free for four months. Then convulsions, vomiting and "temporary" aphasia. Well for three years. Convulsions, headache, vomiting, meningitis and death. No autopsy.

Carpenter, E. R.: Pneumoventriculography in the Localization of Brain Abscess. *Arch. Oto-Laryngol.*, p. 392 (April), 1925. Report of two cases. Case 1.—Male, age 44. O. M. P. A. Acute right frontal sinusitis. Endonasal drainage of frontal and antrum. Intense headache. Vomiting. Indistinct speech. Stupor. Vision blurred. Weakness of left arm, leg and face. X-ray showed cloudy right frontal and right mastoid. Marked left Babinski. Bilateral choked discs. Pneumoventriculography revealed right frontal lobe abscess. Verified by operation. Death. Case 2.—Boy, age 12. History of acute left frontal sinusitis. Headache and swelling over left eye. Spinal fluid cell count 2,400. Abscess found over frontal. Opened and drained two weeks. Slow pulse, vomiting. Osteomyelitis, frontal bone. Thrombosis of superior longitudinal sinus. Operated upon. Carpenter took charge after this history. Could not walk. Headaches. Vomiting. Paralysis right external rectus. Marked Babinski on right side. Right patellar reflex lost. Bulging operative wound. Pneumoventriculography showed right frontal lobe abscess. King operation. Improvement for six weeks. Convulsion, vomiting and unconsciousness. Old wound reopened and pus evacuated. Recovery.

Boss, L.: Diagnosis and Treatment of Rhinogenous Frontal Brain Abscess. (Report of two cases.) *Ztschr. f. Laryngol., Rhinol., etc.*, 13, 128-135, 1924-25. Case 1.—Acute empy-



ema, right frontal. Radical Killian. Pus and polypoid mucosa. Fistula into left frontal through septum. Left radical Killian. Anterior wall perforated. Improvement. Headache and vertigo. Bilateral choked discs. Both frontals opened. Right contained pus. Dura exposed on right and puncture negative. Dura on left exposed the following day, and puncture of frontal lobe evacuated 15 cc. pus. Patient's condition improved, and then symptoms returned off and on for one year. Right frontal lobe punctured three times and no pus obtained. Left frontal lobe punctured or drained seven times. Pus obtained at each operation. Finally meningitis and death. Multiple abscesses of left frontal lobe. Meningitis. Two healed cysts with thickened walls. Case 2.—Acute right frontal sinusitis following an automobile accident. Ethmoidectomy. Week later, radical Killian. The wound reopened later and some bone removed. Posterior plate normal. Headache and vomiting. Posterior plate taken down. Dura purple, and covered with fine fibrin. Dura punctured and two teaspoonfuls of pus located at a depth of one cm. Incision and drainage. At the time of his report, eight days after operation, patient appeared to be on the road to recovery.

Parrish, Robert E.: Report of Two Cases of Brain Abscess Present in Patients Suffering From Nasal Accessory Sinus Disease. *Laryngoscope*, XXVI, p. 336, 1926. Case 1—Male, age 26. Pansinusitis. Operation, subdural abscess secondary to ethmoiditis, localized over fissure of Rolando. Parrish saw the patient three and one-half months after operation. Hernia of brain through trephine opening. Right frontal sinusitis and empyema right antrum. Paralysis of left side. Death. Autopsy. Abscess right cerebral lobe. Culture, nonhemolytic streptococcus. Case 2—Male, age 34, colored. Blood Wassermann + +. Vision failing for six years. Paralysis of right internal rectus. Simple white atrophy of right eye. Pus discharging from right nostril. Caldwell-Luc operation right antrum. Epileptic seizure, coma and death. Autopsy. Ethmoiditis. Dura dark over ethmoid area. Right frontal lobe abscess. Culture, *B. pyocyaneus*.

Jessaman, L. W.: Abscess of the Frontal Lobe, Secondary to Suppuration of the Frontal Sinus with Report of Case;

Boston M. and S. J., CXCII, p. 739, 1925. Male, age 39. Acute right frontal sinusitis, right anterior ethmoidectomy and partial turbinectomy; convulsion that night; drowsiness; radical frontal operation (right); pus; necrosis of septum between sinuses. Posterior plate appeared healthy; posterior plate trephined. Dura dark and under tension; dura and cortex incised. Abscess located and evacuated. Limiting wall. Recovery.

Tarasido, A: Acute Frontal Sinusitis; Orbitopalpebral Phlegmon; Cerebral Abscess, *Rev. Soc. Argent. de Otorrinolaringologia*, 1:23-26 (June), 1925. Male, age 21. Acute right frontal sinusitis. Orbitopalpebral phlegmon. Radical external frontal operation. Pus. Ethmoids removed. Chills, intense headache and temperature. Spinal fluid turbid. Reflexes decreased. Posterior wall trephined. Area of plastic meningitis. Puncture and pus. Incision and cannula located abscess at depth of seven centimeters. Meningitis. Death.

Salinger, Samuel: Frontal Lobe Abscess; Subsequent History of a Case Previously Reported. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, XXXIV, 210, 1925-1926. Male, age 18. In June, 1924, reported as a recovered case of frontal lobe abscess following frontoethmoidal disease. Symptom free three months. Returned for plastic operation. Fifteen days after discharge from hospital, returned. Headache and vomiting. Small opening made in scar and pus evacuated. Rigidity of neck and positive Kernig. Old wound reopened. Pus under frontal bone. Incision into old abscess cavity. No pus. Death. Autopsy. Secondary abscess encapsulated, well back of old cavity, and did not communicate with it.

Cusatelli, A.: A Case of Abscess of the Frontal Lobe of Sinus Origin and a Cerebral Cysticerosis, *Osp. maggiore*, 14:143-147 (May), 1926. Woman, age 21. Occasional loss of consciousness without convulsions since nine years of age. Fell during an attack and struck head, producing lacerated contused wound at root of nose and left supraorbital region. Pain and edema continued. Headaches, temperature and chills. Stupor. Knee jerks exaggerated on right. Pus in nose. Supraorbital wound discharging. Radical frontal operation. Small perforation, posterior plate. Normal dura. Puncture

of frontal lobe and 40 cc. of pus evacuated. Incision and drainage. Death. Autopsy. Meningitis. Large abscess cavity communicating with lateral ventricle. Small cysticerci found over surface of brain.

Motley, F. E.: Acute Brain Abscess as Otorhinologic Complication with Report of Cases. *Southern M. and S. J.*, LXXXIX, 243, 1927. Boy, age 10. Swelling and redness, left orbit. Headache and temperature. Pulse slow at times. Nephritis. Radical frontal, ethmoid and sphenoid operation. Pus and necrotic mucosa in sinus. No perforation of posterior plate. Spinal fluid showed one hundred cells (polys) and cocci free in fluid. Recovery. Seven weeks later reoperated for osteomyelitis of frontal bone (outer table). Healed. Eight weeks later, nausea and vomiting. Bilateral choked discs. Decompression operation. Frontal lobe punctured. Pus one centimeter under surface of brain. Abscess with limiting wall. Mosher brain drain used. Culture, staphylococcus. Recovery.

Arnoldson, N. and Bostrom, C. G.: Cerebral Abscess of Rhinologic Origin, *Acta. Otolaryngol.*, Stockholm, 8:339, 1925. Boy, age 14. Acute suppurative left frontal sinusitis. Headache and tenderness. Edema and vomiting. Endonasal, frontal and ethmoid drainage. Pain, vomiting and apathy. Pulse, 60 to 64. Bilateral papillary stasis. Increased spinal fluid pressure—22 mm. Cells, 22 to field. Radical frontal operation. Pus and granulation tissue. Posterior plate appeared healthy. Dura discolored and under tension. Abscess the size of an egg in frontal lobe. Culture sterile. Recovery.

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LXXVII.

SOME CLINICAL OBSERVATIONS ON THE  
EUSTACHIAN TUBE.\*

BY DUNBAR ROY, M. D.,

ATLANTA.

My object in presenting this paper is for the purpose of bringing before you no new and startling facts about the eustachian tube but to try and lead other younger men in the profession to study along lines which are only suggestive on my part but if carried out to a legitimate end may be productive of clearing up certain points on the obscure subject of deafness.

The rôle played by this tube in the cause of deafness may have been overestimated by some and underestimated by others. Ever since the study of otology began, students and writers in this field have been led to consider some anomaly of the eustachian tube as the cause of deafness in the largest number of people afflicted with this condition. Possibly this may be true. Certainly an altered function of this tube plays a no inconsiderable part in aural deafness.

As a carrier of infection or inflammatory material from the nasopharynx to the middle ear, this tube plays an important rôle.

Why is it that among the large number of individuals suffering with acute attacks of nasopharyngitis or an acute rhinitis, only a limited number have an infection in the middle ear? Is it a peculiar immunity existing in such individuals or is there some inherent quality in the anatomic or physiologic structure of the tube itself?

Does the ciliated epithelium lining the lumen of this tube prevent the passage of infectious material into the middle ear?

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Does not the movement of the cilia have a tendency to take the secretion away from the middle ear?

Why is it that an acute otitis media occurring in a patient apparently not suffering from a sore throat or a cold in the head is so frequently more severe in character than those where such a relationship exists?

Do the walls of the tube remain in contact or is there a distinct opening throughout its length?

Certainly these are questions which could be intelligently studied by the younger otologists.

Hyrtl, the great anatomist, wrote, in 1845: "The walls of the convoluted trumpet are supposed to lie upon each other and form mucous adhesions, so that, as anyone can easily satisfy himself, a considerable degree of compression of air in the mouth and nose is necessary to force air into the *canalis tympani*." If this be true, and it cannot be denied, is not the incorrect blowing of secretions from the nose the cause of 90 per cent of the infections in the middle ear? Should not the proper manner of blowing the nose be taught in the schools and be as important a part of the curriculum as the teaching of other hygienic measures?

To my mind, one of the greatest otologists who ever lived was Sir Joseph Toynbee, and his textbook on otology is still a classic, although published in 1860. The wealth of clinical and pathologic material contained in this book is marvelous, and but few of his deductions have ever been altered. For instance, his anatomic findings were based on 1,523 dissections of the eustachian tube. What otologist of the present day would have the time or material to undertake such a stupendous work?

If we will examine the findings of various anatomists and otologists, we will see that there is some diversity of opinion in their statements.

For instance, Toynbee says that the whole length of the eustachian tube is from one and one-half to two inches, or about 38 to 50 millimeters. Politzer, quoting Bezold, gives this measurement about 34 to 36 millimeters—quite a difference. Is this not probably due to the races of people examined? Both agree that in children the tube is much shorter

and larger in caliber than in adults, hence the greater frequency of middle ear inflammation in the former.

Nearly all writers on this subject agree that the lumen of the tube is practically in contact except when air is forced through into the middle ear. In the cartilaginous portion Bezold says it is three millimeters deep and only one-fourth millimeter wide—a mere slit. I believe this width is an error unless the lining mucous membrane is capable of great stretching.

To the writer's mind, there is much doubt as to the walls of the tube lying in contact. If such were the case, there would exist too much effort for the full ventilation of the middle ear. How often with an acute rhinitis do we feel a stopping of the tube after blowing the nose but which opens freely immediately afterwards without any active effort on our part. If the walls were in constant contact, every little congestion about the upper respiratory tract would cause a feeling of fullness in the ears. Then again, inflate a normal tube with compressed air through the catheter and see how tubular are the sounds heard through the diagnostic tube.

The bony portion, according to Bezold, is two millimeters wide, but does not state its contour. He also states that at its opening into the middle ear it is four and five-tenths millimeters high and three and three-tenths millimeters wide—certainly much larger than most of us imagine.

Toynbee, in 1853, in a paper before the Royal Society of London, endeavored to show that in repose the faucial orifice of the eustachian tube is always closed and that it is opened during the act of deglutition by the tensor palati and levator palati muscles. This statement is almost universally accepted. The value of this observation has not received the clinical consideration which it deserves.

Weakness of these muscles will in time so keep the mouth of the tube closed that stenosis and lack of ventilation may encourage obstructive conditions. All of us have, no doubt, noticed the peculiar guttural voice of deaf people, as if the palatal muscles were undeveloped. Hence some of the benefit derived by massage in the region of the tubal opening, practiced with some degree of success by the so-called osteopathic otologists.

I know of no better classification of eustachian tube obstruction than that given by Toynbee.

1. At its faucial orifice, a thickening or relaxation of mucous membrane or inactivity of the palatal muscles.

2. At its tympanic orifice, thickening of the mucous membrane or deposit of fibrin.

3. In the middle part of the tube a collection of mucus, a stricture of the osseous or cartilaginous portion, or membranous bands connecting the walls.

Does obstruction of the eustachian tube play as prominent a part in the production of deafness as the older writers considered and is still held by many otologists of the present day?

Personally I believe that this pathologic condition has not been sufficiently considered in the last few years on account of the prominence which has been given to otosclerosis. This is a broad field for discussion, and I would not attempt such a discussion in a paper of limited extent.

From my own observations I believe that stenosis of this tube plays a prominent part at the beginning of all deafness of the so-called progressive variety. However, I am also convinced that in a short time after the beginning of deafness there arise other pathologic changes closely related to certain hereditary and systemic diseases and peculiar sclerosing conditions of the mucous membrane in the tube and the middle ear. If such were not the case, then all cases of defective hearing would improve just as soon as the eustachian tube was permanently opened. We know that this is not true.

However, the writer feels that not enough attention has been given to the relief of this tubal stenosis, the otologist being too content with ordinary inflations and the removal of nasal obstructions, together with the adenoids and tonsils.

With this preliminary, I wish to discuss for a few minutes my clinical observations as to which are the best methods for the relief of this stenosis.

Bear in mind the classification of Toynbee.

In the inflammatory swelling of the tubal mucous membrane, accompanied by pain the middle ear and congestion of the membrana tympani and which frequently passes into a discharge through the auditory canal, the writer has often been able to stop this process by the application of a 2 per cent solu-



tion of nitrate of silver to the tubal lining. This is done by cotton on the end of wires, passed through the catheter and as far up into the tube as possible.

Others have written in regard to this method, but I wish to call attention to its distinct value. I feel sure that this procedure may prevent a future stenosis and will aid materially in the rapid return of normal hearing.

In considering a chronic tubal stenosis we have to deal with a different problem. In order to draw conclusions as to the condition of the lumen of the eustachian tube only two methods are valuable.

The faucial opening of the tube should always be inspected either with a postnasal mirror or the nasopharyngoscope. Much valuable information is frequently obtained from this source. As to the tube proper, the diagnostic tube (incorrectly called otoscope) and the eustachian bougies will give us our most definite information.

The use of the proper catheter has much to do with the success of these methods in addition to compressed air through a cutoff which fits tightly in its free end. The writer has not used the Politzer bag in his office for many years.

In reference to bougies, I find myself using them more frequently and in larger sizes.

For a long time my conception of the size of the lumen of the eustachian tube deterred me from using any bougies except the smaller sizes. Now I use much larger ones and find that I get much better results and they are more easily passed. I have also found that bougies of stiff fiber are much the best, as rubber bougies will not give the proper leverage to push them through the strictured points. Then again, I try to have them remain in situ for an hour and pass them daily if necessary.

About fourteen years ago, in Chicago, before the Otological Section of the American Medical Association, I read a paper on "Nasal Analgesia as a Prognostic Symptom in Progressive Deafness." It produced some discussion, but evidently there were few who considered it of any value. Since then I am even more convinced of its material value as an aid in prognosis.

Now I find that the analgesia also extends to the eustachian tube and that the passage of bougies in individuals suffering from progressive deafness rarely produces much discomfort in its passage. The less discomfort or the less sensibility in its passage, the less favorable is the prognosis.

The writer wishes to urge those who have not sufficiently considered the stenosed tube and who have not used bougies to any great extent to give this method a new trial, even if other pathologic conditions should exist.

I have in the last few years seen some excellent results where a marked stenosis did exist and where a gradual dilatation was successfully used. It takes persistence on the part of the otologist and the patient, for the occasional passage of the bougie and its retention for one or two minutes is not sufficient to produce permanent results.

The passing of bougies is a delicate operation, and if not done easily by the sense of touch it is always best to use the pharyngoscope through the opposite nasal cavity so as to be sure that the bougie enters the tube correctly. I have for two years been injecting the tube with thiosinamin in chloretone inhalant, after which the bougie is passed. Experience teaches all otologists never to inflate the middle ear immediately after the use of the bougie, on account of the emphysema which is liable to occur in the neck. Years ago the writer used metal bougies with electrolysis, according to Dr. Duel of New York. This was discontinued because the results seem better with the fiber bougies.

This paper has presented no new and startling original work, but only the hope that the younger otologists may pursue to a more definite end the suggestions and observations herein incorporated, leading perhaps to more tangible results in benefiting a condition which still remains in much obscurity.

## LXXVIII.

### THE DIAGNOSIS OF SINUS DISEASE BY INJECTION OF OPAQUE SOLUTIONS.

BY H. A. VAN OSDOL, M. D.,  
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Difficulties in accurate diagnosis by means of the clinical methods now at our disposal have been met with frequently, and considerable uncertainty has attended dealing with the sinuses surgically, owing to an imperfect acquaintance with the pathology or the anomalies that are present in not a few of the accessory air cavities or sinuses of the head. It is a matter of common experience that cases of sinus diseases are from time to time met with in which the surgeon is unable to know the condition of a sinus without first carrying out some surgical procedure; if he were able, in doubtful cases, to arrive at a correct diagnosis without such preliminary investigation, it would in many cases prove advantageous to the patient.

The idea of using an opaque solution as a contrast medium to fill the accessory sinuses of the nose for diagnostic and therapeutic control is not a new one; however, the literature on the subject is very scarce, and the application of the methods has not been extensive. A contrast medium of an aqueous mixture of lead sulphate was used by Moritz Weil<sup>1</sup> in 1902-03; in 1919 Beck and Ramdoher<sup>2</sup> made injections with bismuth; a 15 per cent solution of potassium iodide and a 20 per cent aqueous mixture of barium sulphate was used by Brunetti and Filippini<sup>3</sup> and reported in 1924.

In April, 1925, a patient was referred to me for a diagnosis of suspected sinus disease; it was important to verify the pathology suspected by X-ray findings as to the existence of an antral polyp, as the patient was not in a good physical condition to undergo a radical operation unless we could show without a reasonable doubt that such an operation was absolutely necessary. The primary X-ray reading was as follows: "Accessory nasal sinuses: Frontals, medium size and depth. No signs of infection. Ethmoids clear with

the exception of the infraorbital group, especially on the right. Maxillary antra: Right shows a circumscribed density filling the lower two-thirds, which appears to be that of a polyp formation. There is slight lack of air in the left. Posterior ethmoids and sphenoids clear. Nasal blocking is noted."

Not knowing that any investigations had been made as to the use of an opaque substance as a diagnostic means of determining the pathologic condition of a suspected sinus disease, but being informed that the urologists had been using some of the silver preparations with a more or less degree of satisfaction in their radiographic studies of the kidneys, I decided to try a method of injection consisting of a 20 per cent aqueous solution of neosilvol into the suspected antri. The injection, 4 cubic centimeters, was made through a needle which had been passed into either antrum underneath the attachment of the inferior turbinate body. My results were as follows: "Examination shows upper part of right antrum filled with the solution; the lower part, which on the primary plate was filled with a circumscribed shadow, shows no opaque solution, and therefore proves a definite filling up of the base apparently by polyp."

It was decided to wait 48 hours and make another picture. The findings at this time were: "Examination of the right antrum shows it to be entirely filled with the opaque solution and there is a large density on the right and on the left. The walls of the antrum show no definite periosteal changes." This reading proved to me that we had a surgical condition to deal with. A Caldwell-Luc operation was done and the antrum found to contain a luxuriant polypus formation, predominating at the base; much thickening of the mucous membrane was present about the normal opening and interfering with drainage.

After this incident I continued my investigations by injecting other cases. The technic used was the same; using 4 to 6 cubic centimeters of 20 per cent neosilvol aqueous solution, the head in the erect position, and filling the antra with the media by passing the solution through a cannula inserted into the normal or the accessory ostium. This method I have found very satisfactory in most cases, and rarely is it necessary to make a needle puncture through the naso-

antral wall under the inferior turbinate. By injecting the opaque solution through the normal or the accessory ostium I can more readily determine when the cavity is full. The location of the ostium is under direct inspection when using the cannula as a guide, and any overflow can easily be seen flowing down over the body of the inferior turbinate. I do not find it necessary to use lavage before injecting, as the opaque solution disseminates so thoroughly that a very definite shadow is shown on the X-ray plate.

The value of an opaque solution as a diagnostic aid was more forcefully impressed on me a little later in 1925, when another patient was referred to me with sinus symptoms. The primary roentgenologic findings were as follows: "Maxillary sinuses show some deficiency of air and thickening of the bases with suggestion of polyp on the left. The latter, however, was not definite." This interpretation is frequently given to us, and is one that the roentgenologist, regardless of how clever he is at reading his plates, is unable to give us more information about. I am sure the surgeon is just as helpless without the aid of some opaque substance to clarify the picture, or by some surgical procedure so that he may see into the sinus by direct inspection or otherwise, and I am equally sure that no one wishes to subject his patient to any exploratory operation until thoroughly satisfied that such surgery is the last resort to verify a suspected pathologic condition.

In this case I had a second picture made after the method described above. The roentgenologic reading was as follows: "Left antrum is entirely filled with the opaque solution and there is no displacement of the solution by polyp." The findings in the second radiogram proved to me that a radical operation was not necessary, but the removal of a part of the nasointral wall, for ventilation and drainage and subsequent irrigation, was done and the patient's sinus symptoms cleared up, as well as an arthritis which was present.

During the time I was carrying on my investigations with neosilvol, two otolaryngologists, Reverchon and Worms,<sup>4</sup> reported in March, 1925, a number of cases wherein diagnosis had been assisted by the injection of iodized oil into the eustachian tube, esophagus and paranasal sinuses. The medium used was lipiodol, an oil extract from poppy seed having an iodine content

of 40 per cent. Jacques Forestier,<sup>5</sup> speaking before the Radiologic Society of North America at Cleveland in 1925, emphasized that lipiodol was not a solution of iodine in vegetable oil, but a definite chemical compound in which the halogen cannot be detected by ordinary reactivities. Lipiodol is wonderfully well tolerated in the cavities of the body. There is no free iodine in lipiodol, so it does not produce irritation nor induce toxicity as the usual iodine might be expected to do.

Since the introduction of lipiodol into this country I am using it as a contrast medium instead of neosilvol, and I believe it offers more resistance to the roentgen rays; the prospectus of the condition present is more clearly shown. I found the topography of the sinus well presented 48 hours after the use of neosilvol, 20 per cent solution, and apparently little of the solution had escaped.

From the therapeutic viewpoint there is a world of difference between superficial alteration of the mucosa, which can be reduced by simple puncture and drainage of the sinus, and fungoid degeneration, either with or without adjacent otitis, for the relief of which the curette must be employed. In the past there was no sure method of differentiating between these two conditions, except to wait the results of conservative treatment, which often meant that a case already too far advanced would be left without adequate treatment for an extended period. Radiography after the injection of an opaque substance enables one to make the distinction immediately, for the outline of an accessory nasal cavity that is almost intact is absolutely different from that offered by one in which the long continued presence of chronic inflammation has produced polyposis.

When an opaque solution is injected into a cavity where such inflammatory products are present, instead of spreading out smoothly and offering a clear outline to the radiographic rays, it will concentrate in one or more sharply limited areas. When exposures are made from several angles upon such a sinus, it is usually easy to decide quite accurately concerning the nature and the extent of the pathologic process which it harbors, as can be shown by a stereoscopic view.

There is, as yet, but little in our own medical literature concerning the use of this form of diagnosis in paranasal

sinuses. Paul B. MacCready<sup>6</sup> of Yale University School of Medicine reports its use in numerous cases of chronic sinusitis without untoward results. Robert H. Fraser<sup>7</sup>, of Battle Creek, Michigan, in reporting thirty-five maxillary sinus cases before the Southwestern Michigan Triological Society, stated that when iodized oil is used in conjunction with other methods, to which its use is complementary, the pathologic findings are of most important assistance in diagnosis and prognosis.

Goodyear<sup>8</sup> of Cincinnati presented a series of thirty-five cases of sinus diseases injected with iodipin, a solution of 40 per cent of iodine in sesame oil, without irritation to the nasal membrane, and, in fact, with considerable medical value. A. W. Proetz<sup>9</sup> of St. Louis, under the title of "Displacement Irrigation," depends on the position of the head to fill the various sinuses for diagnosis and conservative treatment.

The technic used by the writer has been the same as that employed in the examination and treatment by irrigation of the paranasal sinuses; that is, using a small curved cannula, passing it into the sinus when possible through the normal or accessory ostium of the maxillary and into the normal ostium of the sphenoid. A method I find very satisfactory in a majority of cases where it is impossible to pass the cannula through the normal or accessory ostium of the maxillary sinus is to make a needle puncture underneath the inferior turbinate. The position of the sphenoidal ostium is constant only so far as it always lies in the anterior wall of the sphenoid and in the sphenoethmoidal recess. The distance of the ostium from the floor of the sphenoid sinus varies, but if painstaking, one can in the majority of cases enter it. The direction of the probe I use for sounding the sphenoid will be shown in a drawing. The probe used, also the cannula, are graduated from five to ten centimeters.

For the injection I use one part lipiodol and one part liquid albolin, and have found that 4 to 6 cubic centimeters are sufficient for maxillary fillings, and 2 to 4 cubic centimeters for the sphenoid. The operation is usually done in my office and the patient is sent to the X-ray laboratory; I have found that very little fluid escapes in the interval.

Being enthusiastic about this method of diagnosis, I am still conservative to the extent that no case presenting sinus

symptoms is injected with the iodized oil until the usual clinical methods are used, and a primary film has been made showing that there is a necessity for a differential diagnosis. I do not agree with some of the writers on this subject, that there is some medicinal value in this use of lipiodol solutions. It is a definite chemical compound and not iodine with an oil base.

However, I did find that with the use of 20 per cent neosilvol there was a shrinkage of the polypoid membrane. It is my opinion that if there is any therapeutic effect from its use, it would only be in those cases in which drainage was very slow, such as in the case in which the solution used is shown by radiogram plainly ten days after injection. In a case of that type I want to establish drainage at once and not wait to see if I am going to get any results from drug therapy; I believe the reason for the retained solution was due to the extreme thickness of the lining mucous membrane. This thickening, which does occur in sinuses, is my reason for injecting the sinus rather than to depend on the displacement method practiced by Proetz. I do not believe it quite so important to know the exact pathology in frontal and ethmoidal sinus infections, as I have observed in many cases where there was proper care given to the maxillary and sphenoidal sinuses, that the frontals and ethmoids had a chance to drain and that the pathologic changes which had taken place had been due to and perpetuated by the pathology present in the larger sinuses.

I have seen no untoward effect from the use of neosilvol or lipiodol, and I believe not a few patients have been saved the experience and time of undergoing a radical operation. I also believe that in some of our obscure focal infections we can, by the use of an opaque solution and the X-ray, locate the pathology in one or more of the accessory sinuses, because not all diseased sinuses, even though they contain polypi and cysts, show a great amount of discharge. I believe there is more hyperplasia of mucous membrane caused by some of the mucopurulent conditions than by pronounced inflammatory suppurations, and the use of an opaque solution in conjunction with the X-ray will verify the diagnosis of this hyperplastic state.



I am indebted to Drs. Cole, Beeler and Smith for their coöperation and aid in the roentgenologic study of these cases.

#### DISCUSSION.

DR. R. C. BEELER, Indianapolis (by invitation): I wish to thank the members of the Academy for the opportunity to discuss a subject which is so important to the roentgenologist. Dr. Van Osdol has given us a comparatively new method; it will afford more help in diagnosing sinus disease than we realize at this time. Very little is found in the literature on this subject.

For a long time the X-ray has shown densities and circumscribed shadows in nasal sinuses, more especially in the maxillaries, that were not clear to or explained by the otolaryngologist. We described them on the X-ray report and drew conclusions. With the majority of you men our reports were not accepted. Some endeavored to help us by making exploratory punctures, but the washings would return clear; there would be no pus. Some even used a black bowl, but they would find only a few shreds. The final conclusion would be that the antrum was negative for infection. Transillumination would fail to show the shadows found on the X-ray film, because the antrum often contained a normal amount of air.

With the method described this morning I would warn radiologists to be careful about their X-ray diagnosis. It would be better to describe the densities that are abnormal, explain them, and make conclusions as to possible causes for them. To prove up abnormal conditions it is necessary to use the opaque injection. Dr. Van Osdol has given us a splendid paper, and his method is a valuable aid to the differential diagnosis of obscure sinus disease, particularly in the sphenoids, where we have had trouble in making a diagnosis. Radiologists have been of help in finding sphenoidal disease, during recent years, thanks to the work of Pfahler, Bowen and Granger.

We must now work out a standard technic; experience will teach us interpretation. At the present time we are not certain of some of our findings. It is necessary to make very rapid exposures if we wish to show the slight thickening in the small cells and in the sphenoid. The least

movement on the part of the patient will cause a failure. Positions must be standardized and serial films made. The intraoral film as described by Pfahler, the Granger position, and lateral views with the head in the position described by Proetz, we have found essential in showing the sphenoids. The solutions used have been neosilvol 20 per cent, and lipiodol 50 per cent; both are satisfactory for roentgenography. The solutions do not run quickly out of the sinuses as one would expect, so the cells may be injected in an office and the patient sent to an X-ray room some distance away. Defects found in the shape of the sinus, as outlined by the opaque solution, undoubtedly mean pathology.

A primary film should always be made. Where there is a suspicious density in the sinus, inject it to prove the presence of pathology. The injection method, I think, is more valuable than the method of Proetz in showing pathologic conditions. By the latter method the sinus is not so well filled as it is when injected. The injection does not afford knowledge of whether or not a sinus ostium is obstructed unless it is followed up days afterwards; an obstruction is demonstrated by retained opaque solution. The injection will show the size of a polyp, its location, and often its shape. Localized abscesses, mucocoeles, exostoses and malignancies will be more definitely diagnosed by the injection method.

In a recent bulletin from a large clinic I notice that they are allowing densities in the nasal sinuses to go undiagnosed. If they would use injections of an opaque solution in these cases, they would find that they are now overlooking a lot of pathology that would be of value if known. Sinus densities on the X-ray film should be explained. The injections of opaque solution will bring closer coöperation between the rhinologist and the roentgenologist, and will lead to a positive diagnosis in many cases of obscure sinus disease.

DR. JOHN W. CARMACK, Indianapolis: The idea of injecting sinuses through punctures and of filling them entirely is good, because the sinus which we want to find out most about is frequently one into which we cannot get solution by the Proetz method. We have had some of these done when I believed there was pus in the sinuses, and it was demonstrated later by washings. By injecting the fluid

before washing we can detect the filling defect due to accumulated pus. When we wash out a sinus, considerable care and time are required in order to be sure that all of the fluid is out of it; we must have correct postures so that all of the fluid may be removed.

There is another point that we regard as significant: when we fill a sinus and the fluid is retained, an obstruction of the normal ostium exists. That has been a useful point to me. We realize that the consistency of the fluid, as determined by its temperature, has something to do with that, but we have been looking with suspicion on those sinuses in which fluid remains although the patient is moved into different positions.

DR. LAFAYETTE PAGE, Indianapolis: My own experience with this method has been limited but I wish to congratulate the essayist on his splendid work. There is no point that is more obscure in the diagnosis of sinus disease than the exact location of the sinuses. This method has been a great help in the diagnosis of lung disease, especially of bronchiectasis and lung abscess. We have used it in a number of cases of lung abscess with great benefit, by injecting lipiodol through the bronchoscope after having aspirated the lung cavity. As a diagnostic method it is invaluable. Heretofore the X-ray has not been satisfactory in giving definite information in these cases; when the report came back we were still in doubt. From the little I have used this new method, and from what Dr. Proetz showed us in Indianapolis a few weeks ago, I realize what can be done. This is a step forward in the diagnosis of many difficult cases, and Dr. Van Osdol has showed commendable acumen in his study and in the presentation of this subject.

DR. ROBERT H. FRASER, Battle Creek, Michigan: Dr. Van Osdol's paper is a most valuable contribution. My appreciation of the opportunity to meet with the Academy members is equaled only by the satisfaction of being able to endorse the essayist's methods and conclusions from our own experience. The chronic and latent type of case which we meet in institutional work often makes recourse to extensive roentgenography necessary, and by the injection method more frequently.

The displacement method of Dr. A. W. Proetz, which, in our own work, because of an independent development of its principles and the convenience of the term we call suffusion, is advised for the upper sinuses. One should begin in the less diseased case in which the ostium requires no shrinking, and the diagnostic effort is rather to exclude than to define a pathologic lesion. Opaque studies have a definite field in the demonstration of thickened membrane which the primary roentgenogram misses. In the classification of more advanced cases, based on their pathologic anatomy, the service is great, and we feel that our diagnosis is at last worthy of and even more dependable than our list of therapeutic measures. Cases capable of spontaneous resolution may be observed accurately as to progress, and any influences operative over a period of time may be judged, with the collection of statistics. A guide is offered as to the type of operation indicated and the direction of approach and drainage. Pathologic material which is to be removed is delineated.

There are certain sites of election for the measurement of diffuse thickenings of the mucoperiosteum to which we refer by the Greek letter mu. By this means, and by the estimation of per cent filling defect, the roentgenologist may report the physical changes without risking the use of the diagnostic names for diseases, in a report which does not in itself establish the diagnosis. The otolaryngologist should make the fillings, and the roentgenologist should then be responsible for the roentgen examination and its report. A short protocol of the steps in injection or suffusion should be supplied to the roentgenologist.

The majority of the earlier observers made only incomplete fillings by injection, which is a weakness, as Dr. Van Osdol shows. We have assembled a set of instruments\* which we like for injection, both by puncture and by the natural ostium, and which are easy to handle and are equipped with the Hurd bayonet lock. For the maxillary, puncture is preferable in the majority of cases, and in these the inferior meatus is usually chosen as the route. A short (6.5 cm. shaft) Lichtwitz needle of small size causes little trauma;

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\*Description to be published in Archives of Otolaryngology.

it is used with a drilling motion and careful palpation through its lumen with a stylet. This length permits it to remain in situ during roentgenography if it is to be used afterwards as an evacuation aid.

DR. H. A. VAN OSDOL, closing: I am very glad, indeed, to hear the discussion. I did not give the history of this case because I did not think it necessary to this paper. However, the pathologic findings might have been important.

I do not believe that it is necessary to always irrigate sinuses, because there is a great deal of work which may tire the patient, and I do not like to blow air into the sinuses anyway. We do not, as a rule, irrigate sinuses and then blow air into them. We make the injection and have a plate made, and probably in from 24 to 48 hours have another made to see whether the solution has displaced the medium we had placed in the sinus. I think it will be quite important to have statistics. I want to thank Dr. Fraser for discussing the paper.

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LXXIX.

THE "SILENT" TONSIL: ITS RELATION TO  
ALBUMINURIA.

BY CLARENCE PORTER JONES, M. D.,

NEWPORT NEWS.

In 1924, we reported nine cases as having been relieved of albuminuria by removing diseased tonsils. All these cases occurred in the year 1923.

In May, 1927, we reported 13 additional cases occurring during the year 1924, which we felt was of some value as a most intensive study was made in each case, and the fact that all happened to be alive and albumin free at that time, over two years having elapsed.

We now wish to add 17 cases to this list, occurring during the year 1925, each case being kept under observation for 12 months, remaining albumin free.

It is quite commonly conceded by physicians that albuminuria may occur in almost any acute disease, especially in exanthematous fevers of childhood. Even frank nephritis is not an unusual complication of acute infections; in fact, it occurs quite frequently after scarlatina, diphtheria and other diseases. However, physicians have not, as a rule, given general recognition to the possibility of renal changes in nasopharyngeal infection, especially tonsillitis. In 1910, H. W. Loeb directed special attention to the fact that acute nephritis is a frequent sequel of tonsillitis, although this is often overlooked by the majority of practitioners. He made a very careful study of the available literature at that time, recording Leyden's observation (1891) of the possibility of nephritis after angina, and Thouvenet's reported case (1894) of marked albuminuria ten days after the onset of acute lacunar tonsillitis. John Lovett Morse, Emil Mayer, Herrick, Friedrich, Mueller, Curschmann and numerous other authors had recognized the occurrence of nephritis after simple acute tonsillitis.

Adler has even recorded the statement that a form of nephritis occurs in at least 75 per cent of all cases of pure tonsillitis.

Frank Billings recognized clearly the etiologic relation of nasopharyngeal infections to arthritis and nephritis. This subject has been emphasized also by Shambaugh, who stressed particularly the possibility that tonsil stumps, however small, may be productive of nephritis and rheumatism. Smith and Baily reported three cases that demonstrate the close relationship between tonsillar and renal infections. In the presence of chronic disease of tonsil with acute nephritis or pyelonephritis, they recommend immediate tonsillectomy for the purpose of preventing permanent organic injury. Cautly has cited two cases. One of his patients was observed for almost three years, after decapsulation of one kidney had been performed because of persistent albuminuria, anasarca and ascites. Subsequent transitory improvement was ascribed to the operation, but recurrence took place, with edema of the legs and massive albuminuria. This "flareup" was ascribed to septic tonsils. Within a month after enucleation of the tonsils the albuminuria disappeared and the patient subsequently enjoyed good health. Bumpus and Meissner report: "It is our custom to have tonsillectomy performed in all cases in which the urinary infection may be reasonably believed to be of focal origin. Since we have adopted this procedure, a surprisingly large number of apparently negative tonsils have been found to hide deep seated virulent infections." Herman C. Bumpus points out that the deeply buried tonsil is the most dangerous, as absorption from the crypts is easy; that the large tonsil, although infected, drains its noxious exudates and bacteria into the throat, which are swallowed to bathe the gastric and intestinal mucosa. Haden declares: "The tonsil may be a focus at any age and should never be eliminated as a possible focus on the basis of negative clinical findings. Infection in sealed off crypts occur very commonly," also, "Acute glomerulonephritis is most apt to come from the tonsil."

Lyman advocates removal of tonsils if a history of previous infection, induration of the tonsil tissue, redness of the anterior pillars or expressible pus. We had the benefit of careful laboratory studies, including urinalysis, in every case of tonsillitis in a large series, in adults as well as children. Imme-

diately after an attack of acute tonsillitis, albumin was found in over 60 per cent of our cases, albumin and pus cells in more than 35 per cent, and albumin, pus cells and casts in over 10 per cent. In the last class of cases there was always a history of more than one previous attack. If tests for albuminuria or for evidences of hematuria were made systematically by laryngologists in every case of tonsillar infection, it is more than probable that Adler's statement that renal damage occurs in 75 percent of all cases will not prove an exaggeration.

#### THE "SILENT" TONSIL.

The mere presence of the tonsils may often be considered a determining cause of renal involvement in nasopharyngeal infection. Because of their general structure, their crypts and recesses, the tonsils offer an excellent refuge for pathogenic microorganisms. Undoubtedly the tonsillar and other lymphoid tissue of the pharynx plays a defensive rôle against local and general infection, but it has been definitely proved also that it serves as a portal of entry for systemic invasion by virulent bacteria. The etiologic relationship of the tonsils to renal disease, including nephritis and pyelonephritis, need not necessarily be evidenced by tonsillar hypertrophy. If diseased, any tonsil or tonsillar remnant, no matter how small, may provoke systemic complications that lead to renal changes. In the final analysis, the kidneys are primarily excretory organs, serving as filters for inorganic salts as well as organic detritus, including bacterial products. The patients often have a septic appearance, are in a general run down condition, and often complain of stiffness in the neck during deglutition. There is usually some enlargement of the sentinel gland. Inspection of the pharynx discloses that the anterior pillar is darker in color than the other pharyngeal structures and is firmly adherent to the tonsil. The surface of the tonsil is more or less smooth, the crypt mouths being very small and difficult to locate. The crypts contain considerable retained secretion under pressure. The tonsil is light in color, for its surface is bathed in a white filmy substance like a very thick saliva. To such a tonsil in a former report we assigned a name, "Silent Tonsil." We see no reason to change the nomenclature. In the presence of this



tonsillar finding, experience has demonstrated that we can confidently anticipate discovering albuminuria.

The 17 cases we report occurred in our clinic at the National Soldiers' Home, Southern Branch. Fifteen are World War soldiers, and 2 women, aged 26 and 31 years. We are deeply indebted to the efficient medical, surgical and technical staff for their invaluable assistance. In no case was there a history of acute tonsillitis of any severity, but each had had persistent albumin for at least four months. Each patient went through the same routine of intensive study, having been examined by men who are most competent in their respective fields, internist, neuropsychiatrist, cardiologist, genitourinologist, surgeon, roentgenologist, etc. A complete laboratory examination, including for malaria, blood sugar and Wassermann, had also been made, as well as complete X-ray studies, with particular reference to teeth and nasal sinuses. Also, in the study of these patients there was an eye single for any suspicion of gall bladder or any gastrointestinal disorder. If there remained in the minds of any of these examiners any suspicion of any additional focus, the case was ruled out. This series would have been very much longer had not such expressions as "suspicious tooth," "shadowy antrum," "heart murmur," "possibly an infected prostate," "doubtful appendix," etc., been made in reports by these examiners. Therefore, in common parlance, these cases "came clean," or as nearly so as it was humanly possible to have them. The urinalysis showed albumin in all cases, pus cells in nine cases, white blood cells in considerable number in six cases, and casts in four cases. After tonsillec-tomy the urine was examined daily for one week, then every third or fourth day, until albumin free urine was found at two more examinations, then again in thirty days. The urine was albumin free, remaining so, in four days in three cases, nine days in two cases, ten days in two cases, twelve days in five cases, sixteen days in two cases, eighteen days in one case, and twenty-two days in two cases. Each had an urinalysis one year later which proved albumin free.

These "silent tonsils" cases were all submitted to tonsillec-tomy. In each case, the general postoperative procedure was identical. The patient was kept in bed for from two to five days, on a light diet with plenty of water and other fluids. On

the day of the operation, thirty minutes before administration of ether, the patient received a hypodermic injection of morphin,  $\frac{1}{8}$  to  $\frac{1}{4}$  grain, with atropin  $\frac{1}{150}$  grain. The tonsils were enucleated without undue haste, extreme care being exercised to remove every vestige of the tonsils, including any sub-tonsillar lymphoid tissue, and all adenoid vegetations. After the operation the patient was kept in bed for three to seven days. During this time urine was examined daily, and an able internist cooperated in attending to the cases. The albuminuria disappeared completely in from four to twenty-two days, and repeated examinations in each case for a year thereafter failed to disclose recurrence of the urinary symptoms. So far as general physical improvement and cure of albuminuria are concerned, the results have been perfect in all patients.

#### CONCLUSIONS.

1. Routine tests will disclose the presence of albuminuria or other evidences of nephritis in the majority of cases of acute tonsillitis or other tonsillar infections.
2. In cases of albuminuria of doubtful origin, careful examination of the pharynx may often determine the etiologic relationship of diseased tonsils.
3. Hypertrophy or severe inflammation of the tonsils is not necessarily an index of the etiologic relationship of the pharyngeal lymphoid tissue to renal involvement. Even small tonsils or a mild attack of tonsillitis may provoke marked nephritis.
4. Thorough enucleation of the tonsils, including the sub-tonsillar lymphoid tissue and adenoid vegetations, assures the prompt cure and prevents the recurrence of the related albuminuria.

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LXXX.

CASE REPORT—MEMBRANOUS LARYNGITIS AND  
TRACHEITIS WITH A STAPHYLOCOCCUS  
AUREUS HEMOLYTICUS INJECTION.\*

By GORDON BERRY, M. D.,

WORCESTER, MASS.

This case is unusual (1) because of the type of infection. At first we recovered streptococci and a staphylococcus aureus with no Klebs-Loeffler bacilli at any time. But when carried further in the laboratory, a staphylococcus aureus hemolyticus was discovered. (2) Because of its manifestation: an abundant crusting suppurative infection of the entire respiratory tract.

Her rhinologist reports no previous chronic nasal or sinus trouble but a mild recurring pharyngitis. After a healthy life, excepting the usual diseases of childhood with recovery, she had passed through two normal pregnancies and was eight months along on her third, under close medical observation. Early in this pregnancy there had been some nausea and of late a somewhat troublesome cough. Four weeks previously her heart and lungs and kidneys were all right, and the blood pressure was 110/76.

On Saturday morning, April 14, 1928, her physician was called for a slight hoarseness and general prostration, the result of a mild recent cold. He found her temperature 100 degrees, her pulse rather rapid, her chest quiet excepting a few stray rales in the left base and no percussion dullness. There was a slight hoarseness, but the pharynx looked normal. He suggested benzoïn inhalations, which she used once without any noted effect. The urine showed urates, a trace of albumen, no casts, no blood. Sunday morning, excepting a few crackling rales at the right apex, the lungs were clear. She was still a little hoarse. The temperature and pulse

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\*Read before the American Bronchoscopic Society, Washington, D. C., April 30, 1928.

were normal and she wanted to get up. Because of the kidney upset, she was told to remain in bed at least another day. That night she called her obstetrician because of a little dyspnea, asthmatic in type. Sensing trouble and thinking labor pains might start up, he advised her to go to the hospital. This dyspnea became rapidly worse.

She was admitted to the obstetric ward at two o'clock that night in great respiratory distress, speaking with difficulty and only in a whisper. Both inspiration and expiration were labored. Morphine and adrenalin subcutaneously and an ephedrine capsule relieved in a measure her alarm and her dyspnea. The temperature was 101 degrees, the pulse 120 and the respirations were 30. The consulting internist found a clear emphysematous lung and advised a laryngologic consultation.

Our examination showed in addition to a crusting pus in her nose and throat, a thick, dry, dirty, brown membrane or crust covering the vocal cords and affording but a small vent in the middle through which she was breathing with difficulty. I went at once to the surgery for some laryngeal forceps with which to remove this obstruction. In my brief absence she became very cyanosed and almost lifeless, but just before strangling to death, and with the help of the attending laryngologist, who pushed a curved hemostat down her throat, she dislodged two large hard casts, about the size and shape of the third digit of a man's little finger. Respiratory relief was immediate, though examination then showed annular crustings still adherent to the tracheal wall lower down.

Though smears showed no Klebs-Loeffler bacilli, 10,000 units of antitoxin were given; without help. Ammonium chloride was given, ten grains every two hours for five doses, and then five grains every three hours for three days. This definitely helped by loosening the secretions.

Labor pains started the first night after admission, and she delivered herself of a live eight months baby. The placental blood and later, a culture from her venous blood, showed no growth. Her temperature dropped to 98 degrees but climbed to 102.6 degrees on Tuesday. That same evening I had to go down for more crusts, first by the indirect

method, then down into the trachea; after which I injected 10 cc. of liquid petrolatum into the trachea, through a straight cannula. On the next two successive mornings I injected 5 cc. of lipiodol into the trachea by the indirect method. These seemed to help the ammonium chloride. After that she was able to dislodge the tracheal casts herself.

On Thursday a pleurisy and a bronchopneumonia were superimposed. The temperature jumped once to 105 degrees, then remained for the rest of her illness around 103 degrees. The pneumonia improved. The membranous scabs were now soft and moist and she was coughing them up fairly readily. Though examination still showed the crusting on the tracheal wall, there seemed to be no respiratory distress right up to the last, excepting what the pleurisy and pneumonia would account for. She was, however, weak. On Saturday the pneumonia involved the other lung and she failed slowly at first, rapidly at the end, expiring Sunday morning, six days after admission and eight days after the onset. Postmortem details are not available for an autopsy was refused.

Membranous bronchial and laryngeal casts in diphtheria are relatively frequent in our major contagious hospitals. By emergency removal of these casts under direct laryngoscopy and by a prompt use of heavy doses of diphtheria antitoxin, these scabs loosen and the hazardous picture clears promptly. But here reliance had to be placed on local lubricants and irritants such as lipiodol with occasional emergency removals of the casts, and on forced doses of ammonium chloride.

The question naturally arises as to whether it was the staphylococcus aureus hemolyticus that caused this membranous manifestation or did the accident of a coexisting pregnancy have any peculiar bearing. I hope other members of this society can shed light on this problem and offer suggestions for its simpler and more efficient handling.

Note: I am indebted to Drs. George O'Day, Oliver Stansfield, Roger Kinnicutt, Joseph O'Connor, John Cahill and Armand Caron for their coöperation in this case.

36 PLEASANT STREET.

PRIMARY TUBERCULOSIS OF THE UPPER  
RESPIRATORY TRACT.

BY FRANK L. DENNIS, M. D.,

COLORADO SPRINGS.

There has always existed a difference of opinion as to whether primary tuberculous invasion of the upper respiration tract ever occurs. This difference seems to involve chiefly a definition of terms. When one man speaks of primary laryngeal tuberculosis, for instance, he means a case in which a lesion elsewhere cannot be demonstrated; to another observer, "primary" means no other antecedent lesion, demonstrable or not. Opinions on both sides of the question might be quoted endlessly. It is striking, however, that almost all who express themselves admit, either tacitly or expressly, the possibility of a primary affection by saying that the cases are so rare that the probability can be disregarded.

Freudenthal<sup>1</sup> insists that "primary tuberculosis of the larynx does occur and more frequently than has hitherto been accepted."

M. Schmidt<sup>2</sup> thinks "tuberculosis can be primary in any part of the upper respiration tract." It must be remembered, however, that he practiced when the present refinements of diagnosis of pulmonary lesions did not obtain. His conception of the term "primary" means no other *demonstrable* lesion, for he says, "By the diagnosis 'primary tuberculosis,' one must always make it clear that no recognized or recognizable tuberculous spot, as originating point, can be present. Practically then, one can speak of a primary tuberculosis when an exact examination does not prove the presence of any other lesion whatever."

He is sure he has seen a number of cases, both tumors of the cords and also ulcers, though they were not proven to be primary by autopsies. He cites in detail a case with two attacks of proven laryngeal tuberculosis extending over a period of

four years. All the while the general health was excellent, and repeated careful examinations by competent men could demonstrate no other lesion. Finally hemoptysis occurred and a right apical lesion was discovered.

Donnellan<sup>3</sup> maintains that primary laryngeal tuberculosis is not so rare. He reviews numerous case reports from the literature, the most convincing one being by B. Frankel, in which there was definite laryngeal tuberculosis with positive sputum and in which autopsy showed the lungs entirely free from evidence of the disease.

Lawrasson Brown<sup>4</sup> discusses Donnellan's paper and questions the conclusions. He thinks "laryngeal tuberculosis rarely if ever a primary disease," and that "the usual physical examination *alone* is worthy of slight consideration." He does not, however, remark on Frankel's case with the negative autopsy.

Shambaugh<sup>5</sup> reported a case of "miliary tubercular nodules involving the nasal mucous membrane, not associated with general miliary tuberculosis." Careful general examination by a competent man revealed no other lesion except a nodule in the left epididymis. He concludes that this latter was the primary focus and that there was certainly no primary lesion in the lungs.

E. H. Rubin,<sup>6</sup> in discussing tuberculosis of the buccal mucous membrane, says: "The primary form is theoretically possible, but for practical purposes this form can be disregarded."

Mitchell<sup>7</sup> reported 106 cases of enlarged cervical lymph nodes in which tuberculosis could not be established clinically; 38 per cent were definitely tuberculous histologically, and 92 gave positive results on animal inoculation. In another series of 100 children with enlarged tonsils, with no sign of pulmonary tuberculosis, but with palpable cervical lymph nodes, 9 per cent of the tonsils were tuberculous.

Spencer<sup>8</sup> has never seen a case of primary laryngeal tuberculosis nor has Carmody.<sup>9</sup> He quotes Arrowsmith, who says: "Primary laryngeal tuberculosis probably never occurs," and George Wood, who thinks it "may be primary."

Dworetzky<sup>10</sup> says laryngeal tuberculosis is *almost always* (italics mine) secondary to pulmonary tuberculosis, and M. D. Brown<sup>11</sup> thinks it possible for oral tuberculosis to be primary, but he has never seen a case in which it was not secondary.



H. G. Runge<sup>12</sup> discusses the question at some length. On account of its rarity he thinks primary laryngeal tuberculosis can be excluded. He evidently doubts that the primary form is a possibility, if the accepted modes of infection are to be believed in. He believes that other parts of the upper respiratory tract, mouth, nose, and pharynx are much more susceptible to infection than the larynx.

Decidedly, there is a lack of unanimity of opinion, with the consensus of belief probably in favor of the idea that laryngeal tuberculosis, at least, is always secondary. Nevertheless, whenever one sees a case where pulmonary lesion cannot be demonstrated, the question is again opened. Such a case is the incentive for this paper.

#### CASE REPORT.

Dr. S. T. R., age 43 years, gives the following history:

Has always been well. In February, 1923, had an ulcer of the tongue. Two months later two others appeared. The first one was excised and pronounced tuberculous by three separate pathologists. Had several Wassermanns, which were all negative. The tongue healed in about eight months under radium and Roentgen ray treatments, and the patient had no further trouble until about a year ago, when he noticed some irritation of the throat. There was little or no pain, no cough, no hoarseness. Several physical and roentgen-ray examinations did not reveal any lung lesion, except in one instance; one of the internists who examined him thought he had a small lung lesion. A diagnosis of laryngeal tuberculosis was made by Dr. Robt. Lynch, and the patient was referred to me on November 14, 1927. He states that he has had pain in the throat for three weeks, no cough or hoarseness.

Examination: Infiltration and ulceration of the epiglottis, aryepiglottic folds and arytenoids. The cords can be seen only in part and do not seem to be affected. His voice is clear. The appearance of the larynx is typical of tuberculosis.

A chest examination and skiagraph by an expert internist was negative for lung tuberculosis. The sputum examinations have so far been negative for tubercle bacilli.

Under heliotherapy and local applications, the ulcers are healing, the infiltration is lessening and his pain is relieved.

## COMMENT.

So far as clinical evidence goes, this is typical laryngeal tuberculosis without lung involvement. There is probably no doubt that the previous tongue ulceration was tuberculous and that the repeated negative Wassermanns and history rule out syphilis. If there be a lung tuberculosis present all the clinical procedures at our command do not reveal it. In spite of all these facts no one can be certain that some small lung focus does not exist. We shall have to diagnose the case, Clinical Laryngeal Tuberculosis, Apparently Primary.

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- 301 FERGUSON BLDG.

THE MANAGEMENT OF MALIGNANCIES OF THE  
ANTRUM, SUPERIOR MAXILLA, PHARYNX AND  
LARYNX AT THE RADIUM INSTITUTE OF  
THE UNIVERSITY OF PARIS.\*

BY GEORGE T. PACK, B. S., M. D.,

BIRMINGHAM, ALA.

At the time of the initial visit of the patient, an immediate biopsy is performed. This is a prerequisite for treatment inasmuch as the authorities believe that even when the diagnosis of cancer is evident, histologic study will reveal information concerning the variety and the radium sensitivity of the tumor in question, which will be invaluable for postradiation and postoperative statistical analyses.

If the tumor has been treated by radium or X-radiation elsewhere, the patient is usually not eligible for admission to the clinic. This is not a hardhearted policy because the Radium Institute is not for immediate charity, but for the purpose of bettering the existing methods of cancer treatment and to establish a rational, scientific basis for radium therapy. This necessitates the primary treatment of the cancer, otherwise the variable radiation dosage used in other institutions, the lack of the original histologic data and the factor of acquired radio-resistance unite to confuse the radium therapist. In the radium treatment of malignancies, if at first one doesn't succeed, one cannot try, try again with any certainty of success.

If the radiation dosage is great but insufficient, or incorrectly timed, or incorrectly spaced, then within six weeks to two months the tumor may develop a radioresistance which persists indefinitely. The röntgen ray is never successful after radium has failed; on the contrary, recurrences after X-radiation are occasionally susceptible to radium treatment.

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When association of the two modes of radiation is purposely planned it is advisable to have the röntgen therapy always precede the treatment by radium.

#### MALIGNANCIES OF THE ANTRUM AND SUPERIOR MAXILLA.

For osteosarcoma of the antrum, surgery alone is the method of choice. But for epithelioma of the antrum, the combination of surgery and radium therapy, as perfected by Doctors Hautant and Monod, is preferred. The X-ray alone is insufficient, but its employment as a preoperative or supplementary measure may be necessary if extensive and multiple extensions from the primary tumor have occurred, as in the case of voluminous cervical adenopathies. It is difficult to sterilize the cancer with radium alone, because of the danger of radionecrosis, prolonged suppuration and cachexia, hence surgical extirpation always precedes the radium therapy.

These antral epitheliomas grow rapidly, become secondarily infected and early invade the nasal fossæ and possibly the alveolar border, the inferior border of the orbit and the malar bone. There are two stages in the evolutionary history of epitheliomas of the superior maxilla:

1. In the first stage the malignant disease may invade the alveolar process, even the nasal fossæ, especially the posterior ethmoid, and the internal part of the floor of the orbit. The skin, lymph glands, orbit and palate are not invaded. Treatment by surgical removal, followed immediately by radium therapy, gives a proportion of 60 per cent cures. The incision is made along the edge of the nose and through the middle of the upper lip to afford sufficient reflexion of the flap in order to permit exploration of the sinuses and correct placement of the radium tubes. As much of the involved bone is removed as possible, followed by curettage of the cavity. Radium therapy is superior to röntgentherapy here, because its action is limited, it does not affect the eye, the radiations are elective and the skin and hair remain normal.

2. In a more advanced stage the skin and lymph nodes may be involved as well as the orbit, the temporal fossa, the palate, the nose and even the intracranial cavity. The results from radium therapy and surgery are not as good as in the preceding class; 33 per cent are cured. The principles of preradiation surgery are ligation of the external carotid artery, fol-

lowed by a very large osseous resection, including the malar bone, the inferior orbital border and, if necessary, cleaning out the orbital cavity. If the skin has been invaded it should be widely excised. The prosthesis is not made until cicatrization is complete, which requires from two to six months.

The radium treatment begins immediately after the operation. The radium tubes are packed in gauze and distributed equidistantly throughout the wound. The time of application of the radium is extended over four days in order to destroy the "mother cells," which divide at successive intervals and therefore at some time pass into the phase of maximal sensibility (law of Bergonie and Tribondeau).

In America, the dosage of radium is expressed in terms of "milligram hours" or "millicurie hours," which is a constant value, being the dose of emission. It is easily computed by multiplying the intensity of the radiation (i. e., quantity; milligrams of radium or millicuries of radium emanation utilized) by the duration in hours of the application. The dosage notation used throughout France makes the dose proportional to the quantity of radium destroyed during the course of its application: this notation (Debierne and Regaud, 1914; Regaud and Ferroux, 1919) is preferable because it is commodious and equally applicable to tubes of radium or of radon. Let us assume that the total therapeutic value of one millicurie of radium emanation is 133 millicurie hours. Therefore, one millicurie destroyed is the equivalent of 133 millicurie hours or of 133 milligram hours. Or one milligram of radium acting for 133 hours may be expressed in terms of dosage as one millicurie destroyed.

In a general manner the dose per unit of surface of the cavity to be radiated is augmented with any increase in the focal distance, augmented with the thickness of the platinum filter, diminished with the extent of the surface radiated, without speaking of the influence exercised on the estimation of the dose by biologic factors, such as differences in radiosensibility.

The average dose is about 20 to 25 millicuries destroyed given by 15 to 20 tubes of radium, each with a platinum wall of one millimeter in thickness for filtration.

The invasion of the cervical lymph glands offers an unhappy and severe prognosis. If the palpable enlargement of these lymph glands persists after the operation, a neck dissection is done and the extirpated glands examined histologically. If the lymph glands are neoplastic, then the entire operated neck region is subjected to irradiation by radium at a distance.

#### MALIGNANCIES OF THE PHARYNX.

Because of their inaccessibility, malignant tumors of the pharynx are impossible to treat by interstitial or topical radium application; the estimation and equal distribution of dosage is a problem difficult to solve. The treatment of choice is by X-radiation, which gives only fair results. The principles of röntgentherapy employed at the Radium Institute are low milliamperage (4 to 6), high voltage (180 kilovolts), heavy filtration and treatment daily over three weeks' time.

The radiosensitivity of the tumors of this area is as follows: (1) The lymphosarcoma is most sensitive to radiation; (2) the ordinary pavement cell epithelioma is least sensitive, and (3) the so-called lymphoepithelioma is somewhere intermediate in sensitivity.

#### MALIGNANCIES OF THE LARYNX (CARCINOMAS).

From the histologic point of view, the epidermoid epitheliomas of the cutaneous type of differentiation are the least sensitive to radiations, but these are nearly always confined to the vocal cords (i. e., intrinsic), consequently can be removed by surgery. If the epidermoid carcinoma is of the mucous membrane type of differentiation, regardless of whether it is intrinsic or extrinsic in location, radiation therapy only should be used. All extrinsic laryngeal carcinomas are treated by radiation alone. The mucous membrane type is more frequently observed on the ventricular fold and in the ventricular cavity, and is quite favorable to röntgentherapy. Clinically the localization of a vascular, exuberant, bulging growth on the ventricular fold seems to indicate great radiosensitivity. Lateral radiographs of the larynx and hypopharynx are routinely made.

There are two principal reasons advanced by the authorities of the Radium Institute for championing X-ray therapy of

laryngeal carcinomas, (1) a negative reason—the inability of surgery to deal with this variety of carcinoma; (2) a positive reason—most of the epitheliomas of this location are relatively radiosensitive. The possibility of curing certain cancers of the larynx by X-radiation has been demonstrated by Professor Regaud and his associates. Their statistics show over 60 per cent of three year cures. A kilovoltage of 175 to 200 is employed; a milliamperage of 4 to 6; a filter of 2 millimeters of zinc, 3 millimeters of aluminum and 2 centimeters of wood to absorb the secondary radiations emitted by the primary filter; a focal distance varying between 40 and 50 centimeters. The maximal number of "champs d'irradiation," or areas, is four—two anterior and two posterior. The "champs," or areas, vary in form (circular or rectangular) and dimensions (45 square centimeters minimum to 130 square centimeters maximum). The total quantity of radiation given is from 57 units Holzkecht to 92 units Holzkecht. According to Dr. Regaud, in order to treat the pavement cell epithelioma with the greatest efficiency, eight to fifteen days should be consumed in the course of treatment.

Certain accidents, such as edema of the larynx, radionecrosis of the cartilages, intense reaction of the skin or mucous membranes may modify the quantity of the dose administered. To prevent these accidents the patient should be prepared, by a preliminary tracheotomy if necessary, or by the removal of foci of infection or the removal of cartilage invaded by the neoplasm. Tracheotomy is done when there is asphyxia or edema of the glottis, but frequently the permeability of the larynx is ameliorated by the first seance of irradiation.

The interstitial use of radium, by the so-called radium puncture method, is never employed in treating carcinomas of the larynx. Sometimes external radium therapy at a distance is employed in lieu of röntgentherapy—in fact, the radium is probably more efficient, but the cost of using it is so high as to prevent its universal employment. From 500 to 750 milligrams of radium, having a filtration of one millimeter of platinum, are placed seven centimeters from the lateral surface of the neck. Distance is obtained by a boxlike wax moulage, which is lined with lead plate 4 millimeters in thickness. The composition of the wax is as follows: Pure beeswax, 100

grams; paraffin fusible at 62 degrees Centigrade, 100 grams, and finely sifted sawdust, 20 grams. The whole mixture is softened readily in hot water at 48 degrees Centigrade and is poured into sheets one centimeter thick. First, the lateral cervical area over the larynx to be radiated is marked out rectangularly with an indelible pencil. Then a sheet of wax while soft is molded to the corresponding neck, shoulder and side of head. On the external surface of this wax is marked an area corresponding to the one delineated on the skin. The lead sheet, 12 centimeters high to prevent lateral radiation, is folded to make a four sided lead wall, which is trimmed to fit over the marked area on the wax. Now without this lead box, molten wax is moulded to give it support and fasten it to the wax moulage of the head, neck and shoulder. Within the lead lined cavity and upon its wax floor are placed upright cork blocks, 5 centimeters high, and upon these as legs is applied another layer of wax, which fits within the lead rectangle. Over this top layer of wax are distributed the radium tubes. The radium is, therefore, seven centimeters from the skin surface. Each side of the neck, facing towards the larynx, receives about 500 millicuries destroyed, which is the equivalent of 66,500 millicurie hours of radiation. This would mean a surface dose of approximately  $3\frac{1}{2}$  millicuries destroyed for every square centimeter of skin surface. Two areas of irradiation only are used, right and left anterolateral. The box is worn from eight to twelve hours daily, depending on the amount of radium available, and the treatment is extended over ten to twelve days.

In some cases of carcinoma of the laryngopharynx, external radium therapy at a greater distance is employed—e. g.,  $8\frac{1}{2}$  centimeters, consequently the dosage for one side of the neck in such a case may be as high as 750 millicuries destroyed.

In the event of palpable and suspicious cervical lymphadenopathies, a preradiative neck dissection is done, followed by careful histologic examination of all the extirpated lymph glands. If secondary or metastatic malignancy is found, then radium is preferred to X-ray, and more intensive radiation is employed.

I wish to express my gratitude to Professor Claud Regaud, director of the Radium Institute of the University of Paris,



and his able colleagues, Dr. Lacassagne, vice-director, Dr. Monod, radium therapist, and Dr. Coutard, röntgenologist, for the information contained herein and for the kindness shown to me during my studies in their institute.

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LXXXIII.

A FATALITY FOLLOWING THE INJECTION OF  
A LOCAL ANESTHETIC.

BY CARL F. SNAPP, M. D.,

GRAND RAPIDS.

Dr. R. H. O'B., age 26, an interne in a local hospital, was scheduled, on December 2, to undergo a tonsillectomy under local anesthesia. He was a healthy, well developed, but slightly slender young man of rather small stature. His present and past history was negative except for a chronic persistent eczema of the hands of some months' duration. He had been examined by a competent internist in an effort to locate the seat of the trouble, but nothing definite could be found except suspicious tonsils, and was referred to the writer for examination of the nose and throat and for a tonsillectomy if found to be indicated. Physical examination was essentially negative throughout except for definite chronic infection of the tonsils.

The patient was in the best of spirits when he seated himself in the operating chair. He had had one cup of coffee and a small slice of toast early that morning, two hours or more preceding the operation. No narcotic or hypodermic of any kind had been administered. The throat was at once injected with a 0.5 per cent novocain solution, to which had been added, just previous to injection, six drops of epinephrin (1/1,000) to the ounce. The novocain solution had been made up fresh two days previously. No cocain was used in any form at any time. The tonsils were both injected in the usual way with four points of injection about each tonsil, approximately two and one-half cc. of the solution being injected at each point.

Immediately upon completion of the injection the patient stated that he felt nauseated. He appeared to be quite pale, and it was thought he was becoming faint. He was told that he would probably feel better in a few moments, and that if we could go right ahead he would soon be back to bed. He seemed to brace up, and the right tonsil was grasped with a

tenaculum, and an incision was just started in the plica beneath the anterior pillar, when the patient gagged and vomited up a small amount of stomach contents into the throat. The tonsil forceps were at once removed, but almost simultaneous with the vomiting the patient began having convulsive movements, became extremely cyanotic and exhibited marked dilatation of the pupils. He immediately went into the most violent convulsion with clonic muscular contractions that threw him practically out of the chair, his body pitching forward into the arms of the operator. Two physicians from an adjoining room immediately came in and assisted in getting him flat on the table, which was let down from the chair position into which it had been placed for the operation. Artificial respiration was hurriedly started, as was also the administration of oxygen, but no sign of life whatever could be obtained at any time. The convulsion lasted about 30 seconds. One cc. of epinephrin (1/1,000) was given directly into the heart muscle with no results, and digifolin was given intravenously.

The syringe which had been used for injection still contained some 2 cc. of the novocain solution. This, together with a sample of the solution from the original container, was sent to the state toxicologist of the Michigan Department of Health for analysis. The following report was returned:

"The solution marked 'Contents of bottle' contained 0.9 per cent of total solids; the solution marked 'Contents of syringe' contained 0.85 per cent of total solids. The close approximate of these results indicates that both solutions are of the same strength. Both solutions contained novocain hydrochloride. I found no cocain in either sample."

A sample from the original flask was also sent to the state toxicologist for quantitative analysis. The following report was returned:

"This solution contains 0.61 grams of novocain hydrochloride per 100 cc. This corresponds to 2.79 grains per fluid ounce."

A skiagraph of the chest was made after death in order to determine the presence of any possible thymic enlargement. This was made by Dr. V. M. Moore, who reported as follows:

"The postmortem examination of the chest reveals no thymic widening. The heart is moderately enlarged. The lung fields are clear."

Fatalities following the use of local anesthesia happen too frequently, as can be readily seen by a review of the literature and by communicating with our colleagues in the established medical centers and clinics throughout the country. The exhaustive report of the Committee for the Study of the Toxic Effects of Local Anesthetics, appointed by the American Medical Association, and published by Dr. Emil Mayer, as chairman, is extremely valuable. (J. A. M. A., March 15, 1924.)

The question arises as to the exact cause of death. Is it, or is it not, the anesthetic? One cannot help but feel that it is not the anesthetic itself, but probably an anaphylactic reaction. Similar experiences have been known to be encountered by the injection of various sera, and even following a hypodermic of sterile water. The element of fear is undoubtedly an important factor, as in a patient who was being prepared for a ligation of the superior thyroid artery and who went into a convulsion similar to the one described above just as the surgeon was filling the syringe with solution preparatory to injection. If this convulsion had come on three or four minutes later it would naturally have been attributed to the effects of the local anesthetic. The patient recovered from the convulsion, however, and the operation was performed with a local anesthetic on the following day without difficulty. (Dr. D. Van Duzen, personal communication.)

The injection of a small amount of the solution as a test and waiting for some minutes for any possible reaction does not seem to the writer to be of any especial value. In this particular patient it was learned later from the family that he had had a molar tooth extracted under novocain some three years previously without any evidence of toxicity.

Two points of especial importance in the further use of local anesthetics were impressed upon the writer by this accident: first, the very careful, cautious injection of the anesthetic, watching closely the general condition of the patient throughout the procedure; and second, the use of the smallest possible amount of solution necessary to control pain. Less than half the amount formerly employed is now being used with equally good anesthesia.

604 MEDICAL ARTS BLDG.

#### LXXXIV.

### COAGULATION TIME BEFORE TONSILLECTOMY.\*

BY MARGARET NOYES KLEINERT, M. D.,

BOSTON.

This report is an analysis of the records of the patients admitted for tonsillectomy and adenectomy, during the last three years, to the New England Hospital. The object of the study was to learn, first, if there is any relation between the patients that bleed and their respective blood clotting time. Second, to make a study of those cases that do bleed in comparison to those which do not, and third, to learn how many bleeding cases there have been.

According to the literature, in spite of the routine taking of the blood clotting time followed in many hospitals, little has been learned from it.

Hunt<sup>1</sup> states that in a questionnaire sent out in 1925 to 567 hospitals, gathering information on the value of coagulation time prior to tonsillectomy with reference to postoperative bleeding, that of the 300 hospitals sending in replies 68 per cent consider a routine coagulation time advisable, 176 hospitals do a routine test, 59 do not, 65 do only when requested, and 91 do not consider it necessary.

Bailey<sup>2</sup> states that in a questionnaire sent to 400 laryngologists, the general opinion was that the routine taking of the blood clotting time was valuable as to laboratory records, but that a careful family history was clinically more important.

Harter<sup>3</sup> reports that from a study of 418 cases it appeared that prolonged coagulation time up to 15 minutes had no relation to hemorrhage; 16 of these cases had profuse bleeding, but 14 of them were under seven minutes coagulation time, one was eight minutes, and one ten minutes.

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\*Presented at the Clinical Conference of the New England Hospital for Women and Children, November, 1927.

## COAGULATION TIME.

In this study 645 records were reviewed. The blood clotting time was found noted 435 times. The omissions were due to privates having had examinations previous to entrance or misunderstanding on the part of the interne as to the necessity of making the test. The tests were made by the interne excepting in a few instances where the hospital technician did it. The capillary tube method was used. The results obtained from the 435 records as to the blood clotting time are given in the table below:

| Coagulation<br>Time | Number<br>of Cases |
|---------------------|--------------------|
| 1 minute .....      | 4                  |
| 2 minutes.....      | 30                 |
| 3 minutes.....      | 164                |
| 4 minutes.....      | 134                |
| 5 minutes.....      | 72                 |
| 6 minutes.....      | 22                 |
| 7 minutes.....      | 8                  |
| 8 minutes.....      | 0                  |
| 9 minutes.....      | 0                  |
| 10 minutes.....     | 1                  |
| Total.....          | 435                |

Of these cases, 298, or 68 per cent, have a coagulation time of three or four minutes. The other 32 per cent consist of 4 at 1 minute, 30 at 2 minutes, 72 at 5 minutes, 22 at 6 minutes, none at 8 or 9 minutes, and there was only one at 10 minutes. The coagulation time longer than six minutes must be comparatively rare, shown by the small number found in this study.

## BLEEDING CASES.

There were five postoperative hemorrhages among these 645 cases. The blood clotting time in those cases that bled was recorded in four instances. In each case it was low, as shown in the table below:

| Age | Hg.  | C. T.     | Bl. Pr. |
|-----|------|-----------|---------|
| 33  | 55   | 4 minutes | -----   |
| 59  | 80   | 5 minutes | -----   |
| 60  | 80   | 5 minutes | 190/102 |
| 55  | ---- | -----     | 112/ 70 |
| 28  | ---- | 5 minutes | 103/ 95 |

There was one clotting at 4 minutes, three at 5 minutes, and one omission. All of these cases were returned to the operating room in from four to nine hours after operation, given an anesthetic and the tonsillar fossæ sutured, tying off bleeding points. There were no complications following the second operation.

## BLOOD PRESSURE.

The blood pressure was recorded in three of the five cases which bled. In the 60-year-old case it was the highest, 190/102. In the case 55 years old it was 112/70, which was slightly low for a woman of 55. The youngest case in this bleeding group had a pressure of 103/95, not remarkable.

## AGES.

Three of the bleeding cases were found to be beyond the usual age of those ordinarily submitting to tonsillectomy, so a table was made to show the ages of all operated upon.

| Age under | 5   | 5-10 | 11-20 | 21-30 | 31-40 | 41-50 | over 50 |
|-----------|-----|------|-------|-------|-------|-------|---------|
|           | 122 | 243  | 127   | 80    | 41    | 21    | 11      |

Four hundred ninety-two, or 76 per cent, were below 20 years of age. One hundred forty-two, or 22 per cent, were between 20 and 50 years old, while 11, or about 2 per cent, were over 50. Of these 11 cases over 50 years of age, 3, or 28 per cent, belonged to the bleeding group.

## HEMOGLOBIN.

One of these cases having postoperative bleeding showed a low hemoglobin, which prompted a study of the records for all cases of 65 per cent or under.

|             |             |             |             |             |
|-------------|-------------|-------------|-------------|-------------|
| No of cases | 17          | 5           | 2           | 2           |
| Hemoglobin  | 65 per cent | 60 per cent | 55 per cent | 50 per cent |

There were 26, or 4 per cent, of the 645 records reviewed with a hemoglobin under 65 per cent. The one case belonging to the bleeding group had a hemoglobin of 55 per cent, a clotting time of 4 minutes, and was 33 years old.

#### CONCLUSION.

There seems to be nothing gained by continuing the routine blood clotting time.

It is important to consider the age, contractility of the tissues and the general physical condition of the patient.

The blood clotting time beyond eight minutes must be comparatively rare, for in this study only one was found to have more than eight minutes' time.

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AVENUES OF INTRACRANIAL INFECTION FROM  
THE NOSE, PARTICULARLY FOLLOWING  
NASAL SURGERY: ALSO RELATION OF  
EXTERNAL INFECTION TO INTRA-  
CRANIAL COMPLICATIONS.

By JOHN A. PRATT, M. D., F. A. C. S.,  
MINNEAPOLIS.

There are six groups of intracranial pathways of infection.

POSSIBLE EMBRYONIC DEHISCENCES.

1. In the embryonic development there are possible ways of leaving a dehiscence that might be a pathway of infection to the meninges. The cribriform of the ethmoid is cartilaginous at birth and does not ossify until the second year. The crista galli ossifies between the second and third years, while the lateral masses, which form the root of the ethmoid capsule, are ossified at birth. Complete fusion of different parts of the ethmoid does not take place until the sixth year and ossification not until the sixteenth year. The ethmoid bone, however, becomes of adult size by the seventh year.

Exenteration of the ethmoid cells on patients under sixteen years of age, with removal of the middle turbinate, would be a very dangerous procedure, but with the middle turbinate left in place, a safe operation.

The basipharyngeal canal, which extends from the sella turcica through the sphenoid bone to the pharynx, is really a part of the embryonic impush of the Rathke pouch and is still open in about 5 per cent of the cases at birth. It could remain open as an embryonic dehiscence from the sphenoid sinus, thus accounting for meningitis from sphenoid infection that leaves no trace of the pathway, especially if the cavernous sinus is not infected.

MIDDLE EAR.

The petrosquamous fissure, which passes through the middle of the tegmentum tympani, might leave an embryonic de-

hiscent pathway to the meninges. As shown by Kisselbach's report of 336 cases examined, the ages ranging from birth to eighteen years, there is a gradual reduction of the closure of this fissure, of from 78.8 per cent to 11.1 per cent. Up to eighteen years of age there is a possibility of eleven out of each hundred having a possible infection through a dehiscence pathway from the middle ear. Thus the meningitis following infections of the nose, throat and ear during early life may be through these embryonic dehiscences.

Obliteration of the petrosquamosal fissure in childhood (summarized from the data of Kisselbach), 336 cases :

| Age—            | No. Cases | —Condition of the petrosquamosal fissure—<br>Course indicated by pores and clefts |                       |                      |                      |
|-----------------|-----------|---|-----------------------|----------------------|----------------------|
|                 |           | Entirely open<br>%  | Partially closed<br>% | Entirely closed<br>% | Entirely closed<br>% |
| Birth to 1 year | 52        | 17.3  | 78.8                  | 0.0                  | 3.8                  |
| 1 to 2 years    | 26        | 0.0   | 46.1                  | 0.0                  | 53.9                 |
| 2 to 3 years    | 46        | 4.3   | 35.6                  | 0.0                  | 52.6                 |
| 3 to 4 years    | 52        | 0.0   | 48.1                  | 17.3                 | 34.6                 |
| 4 to 5 years    | 36        | 8.0   | 31.5                  | 13.2                 | 47.4                 |
| 5 to 10 years   | 86        | 0.0   | 20.5                  | 8.8                  | 70.7                 |
| 10 to 15 years  | 20        | 10.0  | 23.3                  | 6.7                  | 60.0                 |
| 15 to 18 years  | 18        | 0.0   | 11.1                  | 22.2                 | 66.7                 |

2. The pathway to the meninges may follow in any direction by continuity, due to destruction of tissue, from malignant or suppurative conditions of the eye, ear, nose and throat. The radical operation on these parts eradicates the point of infection and tends to prevent the spread of the disease to the meninges.

3. The venous pathways are those taken by infections that seemingly start in the most simple way and lead to the most serious results.

The point of attack preceding the meningeal infection is the cavernous sinus.

The portion of the cavernous sinus first invaded depends on the venous pathway taken by the infection. Dr. Eagleton has named these as follows: (a) the anterior, (b) the anterior inferior, (c) the posterior inferior, (d) the posterior, and (e) the mesial paths.

(a) The current in the veins of the anterior part of the face normally drain into the facial vein and thence into the jugulars. Owing to the lack of valves in these veins, a slight blockage may deflect the current through the communicating angular vein into the ophthalmic vein, then into the cavernous sinuses and the other sinuses of the head.

Infections of the face may, by venous circulation or retrograde thrombophlebitis, cause thrombosis of the cavernous sinus and eventually meningitis. Early destruction of the angular vein would block this pathway. This is called the anterior, or ophthalmic pathway.

(b) The pterygoid plexus receives veins from the deep tissues of the side of the face, the pharynx and the tonsillar region, emptying into the facial vein and then into the jugular. It also has communicating branches which empty into the ophthalmic vein and the cavernous sinus, and by these branches may carry infection into the cavernous sinus. This is called the anterior inferior pathway.

(c) Any infection that would cause a thrombosis of the jugular vein could, by a retrograde thrombophlebitis, follow through the bulb and the inferior petrosal sinus to the cavernous sinus. This is called the posterior inferior pathway.

The infrequent infections of the cavernous sinus from a thrombosis of the lateral sinus, is due to the angle of insertion of the petrosal sinuses. A descending infection from the lateral sinus obliterates these openings, while a retrograde thrombophlebitis of the jugular vein will infect it.

(d) An ear infection that would cause a thrombosis of the inferior petrosal sinus directly is called the posterior pathway.

(e) The osseous veins of the sphenoid wall and the veins of the carotid plexus are also routes to the cavernous sinus. This is known as the mesial pathway.

4. In fulminating meningitis, which is frequently preceded by an acute pneumococci or streptococci otitis, a blood stream infection would affect the prearachnoid spaces of the cortex. It may also affect the choroid plexus of the ventricles, the ventricles and later the meninges. This is the blood stream pathway.

5. The instrumental pathway caused by perforation into the brain through the cribriform plate and false passage by frontal probes needs only to be mentioned.

In 1924 I presented, before the Nose and Throat Section of the American Medical Association the fact that the removal of the middle and superior turbinates in exenteration of the ethmoid cells and opening of the frontal sinuses intranasally might open the sheaths of the olfactory nerves and thus give a direct pathway to the meninges. It was further stated that the majority of deaths from meningitis following operative procedure in this region was due to this cause. Eighty deaths were cited in the private practice of fifty-two of our leading rhinologists, and in seventy-eight of these all or part of the middle and superior turbinates were removed.

June 4, 1926, Drs. Turner and Reynolds of Edinburgh reported the macro- and microscopical findings in a death from meningitis, following an intranasal operation in the region of the olfactory nerves, showing that the infection spread to the brain through the olfactory nerve sheaths. Thus proving my contention of 1924, that there is this pathway through the sheaths of the olfactory nerves.

#### PATHS BY OTITIC INFECTION.

6. There are two pathways of infection to the meninges through the internal ear. One, by way of the perilymph, and the other by way of the endolymph.

The perilymph occupies the space between the membranous labyrinth and the bony labyrinth and the two spiral cavities in the cochlea, called the scala tympani and the scala vestibuli. The perilymph is in communication with the subarachnoid space by way of the cochlear aqueduct, which starts on the floor of the scala tympani near the round window and leads through the petrous bone to the outer edge of the jugular foramen, where the bony cochlear aqueduct ends but the membranous cochlear aqueduct continues through the jugular foramen and perforates the dura. Thus there is a direct pathway to the meninges if an infection of the perilymph is present.

The endolymph, which occupies the membranous labyrinth and the scala media of the cochlea, communicates by way of the ductus endolymphaticus through the petrous bone to the saccus

endolymphaticus, which lies within the cranium on the external third of the posterior portion of the petrous bone. Ulcerative infections of this space would give direct contact with the dura.

Surgical injury of the semicircular canals, cochlea and stapes during mastoid operations would infect these two pathways, as would, of course, general suppurative infections of the ear.

7. There are no lymphatics in the brain, and the so-called lymph spaces drain into the large sinuses.

The lymphatics of the head and neck have no communication with the brain, but all drain away from the brain into the lymphatic glands of the neck and then into the portal system.

In the early stages of bacterial infection the lymphocytes and phagocytes in the lymphatics attack the invader, blocking them at the lymphatic gland. As soon as thrombosis takes place they cease to function.

This protective drainage by the lymphatics is particularly active in the mucous membrane of the eye, ear, nose and throat, showing the cervical gland tenderness in beginning infections before the more serious thrombosis takes place.

#### SUMMARY.

##### PATHS OF INFECTION.

1. Paths left open by embryonic dehiscences, as in the ethmoid bone, the basi-pharyngeal canal to the sella turcica and the petrosquamous fissure from the tegmentum tympani.

2. Paths by continuity of diseased soft tissue and osteomyelitis.

3. Venous pathways taken by infection of the face, nasal cavities, mouth and pharynx, causing anterior or posterior thrombosis of the cavernous sinus by retrograde thrombophlebitis.

4. Direct infection by a septic blood stream.

5. Pathways opened by surgical trauma, giving direct communication to the meninges, or injury to the olfactory nerves, thus opening the sheaths that are directly connected with the meningeal spaces.

6. Pathways of otitic infection.

7. The part played by the lymphatics.

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LXXXVI.

PROBLEMS IN BRONCHOSCOPY AND  
ESOPHAGOSCOPY.\*

By G. W. BOOT, M. D.,

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The first problem the bronchoscopist has to meet is: When is bronchoscopy indicated? This problem is not as important now as it was a few years ago, when bronchoscopy was felt to be more serious than it is nowadays. Bronchoscopy is not a serious procedure under ordinary circumstances, yet it is decidedly more unpleasant than any ordinary examination. If it can be done under a general anesthetic, well and good, but if it must be done under local anesthesia or under no anesthesia at all, the sense of suffocation and the almost irresistible inclination to put up the hands and push the operator's hands and instruments away, together with the most unpleasant stretching of the neck muscles, make it an operation that patients are not willing to repeat. So for purposes of examination and diagnosis, it will never be resorted to as freely as say cystoscopy. However, in certain cases its use for diagnosis should be made use of in spite of its disagreeableness. Such cases will be those where the signs and history point to the presence of a foreign body that does not show in the X-ray picture; cases of lung abscess, gangrene of the lung, bronchiectasis, and the like. Patients not infrequently come to the bronchoscopist with the suspicion of a foreign body in the air passages, and yet the foreign body does not show on X-ray examination. Such cases are apt to have unusual râles, with unusual distribution, that do not clear up on proper internal medication over a reasonable time. They may or may not have fever. Ordinarily a history can be obtained of a severe strangling or choking spell or of a severe cough of sudden onset that lasted for several hours or even days. With such a history,

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\*Read before the Chicago Laryngological and Otological Society, March, 1927.

even in the absence of a shadow in the X-ray picture, bronchoscopy for diagnosis should be done.

When, after a tonsillectomy, particularly if it is done under profound general anesthesia, the patient begins to cough and the physical signs indicate the possibility of a beginning lung abscess, bronchoscopy is indicated for the diagnosis of the possibility of inhalation of some foreign substance.

Another problem in bronchoscopy is whether an immediate bronchoscopy should be done or whether the patient can wait on the convenience of the operator. What cases demand immediate bronchoscopy? Chiefly the cases that have inhaled a foreign body of such size or shape that respiration is considerably embarrassed. Such a case I had recently, where a schoolgirl of eight inhaled her lead pencil. This obstructed the trachea to such a degree that there was imminent danger of suffocation. Other cases are where the foreign body is smaller but movable, and comes up against the under surface of the glottis in such a way as to completely cut off the supply of air.

The ordinary case that has inhaled a foreign body can safely wait until careful and complete preparation is made for the operation, even though it necessitates a wait until the next day or a visit to the metropolis. In most cases an X-ray picture can be made before there is any great need of doing bronchoscopy. It should be done in all cases where it is possible. Peanuts cause so much reaction that I do not advise waiting until the next day, unless it is absolutely unavoidable.

What type of instrument should be used? Each type of instrument has its advantages. The Bruening instruments can be used in smaller tracheæ and bronchi than can the Jackson instruments. On the other hand, the Bruening instruments have to be used largely by the sense of touch, while the Jackson instruments can be used by sight. The Jackson instruments require forceps of smaller diameter, and consequently weaker, than do the Bruening instruments. When it is necessary to cut the foreign body before removing it, the Bruening instruments are to be preferred. A much larger foreign body may be removed through the tube of the Bruening instrument than through the Jackson instrument.



Which shall be done, upper bronchoscopy or lower bronchoscopy? For the beginner, lower bronchoscopy is safer and will be used oftener than upper bronchoscopy. The danger from a tracheotomy is not much if ordinary surgical skill is used. If the foreign body is very rough or irregular, tracheotomy should precede the bronchoscopy. It should also be done if the foreign body is too large to pass through the tube and is of a friable nature so that it can break while passing between the vocal cords. One of the few deaths I have had from bronchoscopy was due to the neglect of this precaution. The foreign body, a large peanut kernel, broke as it was passing through the glottis, and, in the excitement that followed, the patient died before the tube could be reintroduced or tracheotomy done. This patient was being operated on under local anesthesia. When he found he was choking, it was impossible to keep him still enough to reintroduce the bronchoscopic tube, and by the time tracheotomy could be done he had stopped breathing forever.

If a rough or irregular foreign body is dragged through the glottis, so much trauma may be done that edema of the glottis results, necessitating tracheotomy afterwards when it may not be convenient—say in the middle of the night. It is much better in such a case to do the tracheotomy first and avoid the injury to the larynx and the danger due to edema.

What anesthetic should be used? Ordinarily, ether if a general anesthetic is to be used at all. Gas does not last long enough. Chloroform is too dangerous. Ether relaxes, which is very desirable for this work. On the other hand, it promotes free secretion of mucus, which is not desirable; consequently, it is advisable to give a full dose of atropin hypodermatically before starting the anesthetic.

Cocain may often be used to advantage in adults if they are of the phlegmatic type. If it is used, it may be necessary to use it in solutions as strong as 25 per cent. It should be applied to the base of the tongue, the pharynx, the arytenoids and interarytenoid space, and to the vocal cords. After the tube is introduced, cocain should also be applied to the carina, for irritation of this spot at the bifurcation of the trachea into the bronchi is sure to excite cough. If a foreign body has become buried by granulations, touching the granulating spot

is sure to excite cough, and this, too, must be anesthetized. Care must be used that poisoning by cocain be not added to shock in doing bronchoscopy. Because of shock bronchoscopy should not be continued more than twenty minutes to a half hour at most, for the intimate relations of the trachea and bronchi to such important structures as the vagus, heart and great vessels as well as the obstruction of respiration caused by the tube all tend to cause shock. With a small child that can be controlled by force, it is often better to simply wrap the child tightly in a sheet and have assistants hold the child forcibly and then do the bronchoscopy with no anesthetic, rather than add to the risk by giving a general anesthetic. This is particularly true where there is so much embarrassment of respiration that there is danger of asphyxia if any of the accessory muscles of respiration are put out of commission by the relaxation of a general anesthetic. It is rough treatment but much better than a dead child.

What about the foreign body that has lodged in the hypopharynx or in the esophagus? Do not attempt to shove it down blindly by means of the stomach tube. I have seen one case that died because the family doctor attempted this. Instead of shoving it down, he shoved it through the wall of the esophagus into the neck, with the result that an abscess formed that burrowed down into the mediastinum, and although the foreign body was removed by external incision and an attempt was made to drain the abscess, the child died of suppurative mediastinitis.

If the foreign body lodges opposite the suprasternal notch, which is its most common place for lodgment, the problem is to get it without accidentally shoving it down farther. This is especially true of open safety pins. It is often so easy to dislodge a foreign body in its region that particular care should be used not to dislodge it.

Another problem is to find the foreign body when lodged opposite the suprasternal notch. Because of the arching forward of the cervical vertebræ when the neck is extended to introduce the bronchoscopic tube, the tube is in danger of passing so far in front of the foreign body that it is not seen at all or only after repeated efforts. A fold of edematous mucosa is apt to be pushed in front of the lip of the broncho-

scope and thus hide the foreign body. On pushing the tube down, it passes anterior to the foreign body. But this is not always the case. Only this morning I had a child of twenty-two months who swallowed a metal disc six months ago. It lodged at the suprasternal notch. In this case the foreign body had lodged so that the tube passed posterior to it, and it was only when the lower end of the tube was directed strongly forwards that the edge of the disc could be seen and the foreign body grasped.

What can be done if the neck cannot be extended? If the head can be rotated and the foreign body is in the trachea or bronchi, tracheotomy can be done and the foreign body removed by lower bronchoscopy.

If the foreign body is in the esophagus, a pharyngotomy must be done and the foreign body removed in this manner, unless the operator is as fortunate as I was in one case, where a man swallowed a large piece of meat which completely blocked the esophagus. It gave no shadow with the X-ray, but it so completely blocked the esophagus that every attempt to swallow water caused strangling. The patient, an adult man, had a rigid neck due to fracture of the cervical vertebrae several years before. Thinking that if he were anesthetized, I might be able to extend his head enough to see the foreign body, I gave him ether, but without any effect on increasing the extrusion of the neck. Fortunately, on awaking from the ether, he vomited up the piece of meat and thus avoided a pharyngotomy, which was the only alternative.

LXXXVII.

TINNITUS AURIUM: SOME CONSIDERATIONS OF  
ITS CAUSES, WITH SPECIAL REFERENCE  
TO ANALOGIES.

By T. J. WILLIAMS, M. D., F. R. C. S.,

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There is perhaps no symptom more continuously referred to the aurist, nor any problem which offers more perplexities to patient and practitioner alike, than the wide range of subjective ear sounds which are grouped under the general title of tinnitus aurium. A large percentage of all the cases referred to the specialist in otology present this symptom, either isolated or in conjunction with others, and not infrequently is the specialist confronted with marked examples wherein he can find no lesion in the auditory apparatus capable of explaining the symptoms.

Comparatively little attention is given by the profession to this special manifestation, if one may judge by the small amount of space devoted to it in standard text books, and this appears all the more remarkable because there is no irregularity in the hearing apparatus which brings the patient to the otologist more promptly than this. Many people will grow increasingly deaf for years without realizing the extent of their infirmity until they are finally driven to consult the otologist, only by the importunities of their friends, but the victim of tinnitus needs no outside urging. He usually flies for succor early, because the nature of his malady is such as to make his life an intolerable burden, and even to render him fearful of his sanity.

Tinnitus is not a disease *per se*, nor is it a definite symptom of some aural condition which the presence of tinnitus enables us to recognize. Its exact pathology is not definitely understood, and no one has yet been able to point his finger and exclaim, "There is the cause of tinnitus!" Although its etiology is not definitely known, we are accustomed to associate all

subjective ear sounds with some disordered condition of the eighth nerve. G. W. Mackenzie<sup>1</sup> places tinnitus or subjective noises first on his list of symptoms of acute eighth nerve neuritis. "Theoretically," he tells us, "every attack of eighth nerve neuritis should present this symptom," though in practice others often mask it to a considerable extent, and he mentions two cases reported by A. W. Mackenzie where tinnitus continued in spite of complete deafness, until absolute destruction or degeneration of the nerve had taken place. He does not give us any light on the question of whether the relation between tinnitus and eighth nerve neuritis is an organic or a functional one.

A careful consideration of the subject will, however, force us to the conclusion that many other factors enter into the causation of this frequently baffling condition. There appear to be two general classes into which most of our cases of tinnitus can be divided.

First.—Where tinnitus is present without any noticeable diminution of hearing and may be due to disturbance of the function of the auditory nerve center of the labyrinth, or to some abnormal state of the circulation.

Second.—Those cases presenting marked deafness which is caused by a definite pathology in the external, middle or inner ear.

Dan McKenzie<sup>2</sup> gives "a useful practical classification" of the different subjective sounds apprehended in tinnitus as simple and compound. In simple tinnitus, the subjective sound is uniform in character, however variable it may be in loudness. Compound tinnitus consists of subjective sounds which vary not only in loudness, but also in character, several distinct noises being simultaneously audible.

The character of these sounds is variously described by sufferers seeking relief. Sir William Wilde, in his *Aural Surgery*, says: "Persons from the country or rural districts draw their similitudes from the objects and noises by which they have been surrounded, as the falling and rushing of water, the singing of birds, the buzzing of bees, and the waving or rustling of trees; while on the other hand, persons living in town,

or in the vicinity of machinery or manufacturers, say they hear the rolling of carriages, hammering and the various noises caused by steam engines. Servants almost invariably add to their other complaints that they suffer from the ringing of bells in their ears, while old women much given to tea drinking, sum up the category of their ailments by saying that all the kettles in the country are boiling in their ears."

McKenzie has found compound tinnitus in the more serious and obstinate forms of middle ear deafness, often preceding cochlear involvement. He has noted it in otosclerosis and in many general diseases as arteriosclerosis, high blood pressure and Bright's disease, etc. In every day practice it is more commonly the simple form which we encounter, and not infrequently the patient's description will aid us in locating the exciting cause. Hissing or singing, rushing, roaring, or even musical notes usually indicate some irritation of the labyrinth at the point where the nerve terminates. Spasmodic contractions of the salpingopharyngeus muscle attached to the eustachian tube might cause a sound which is described as "clicking" or "crackling"; while the presence of any exudate in the middle ear might be responsible for the complaints of "bubbling noises." Again a "humming sound" may be produced by a clonic contraction of the stapedius and tensor tympani—the intrinsic muscle of the ear. Circulatory disturbances may give rise to pulsating or "beating noises"; for instance, if there is a diminution of vasomotor inhibition, so that the vessels are dilated and contain an excess of blood. It is easy to visualize the nerve endings among which these vessels ramify, conveying the perception of their pulsations to the patient. Likewise, if tension in the blood vessels be diminished, the physical balance between the intralabyrinthine fluid and the vessels will be upset, resulting in the perception of a faintly pulsating tinnitus. This form of tinnitus could easily occur in an individual who exhibits no organic disease.

#### ANALOGIES.

Consideration of the eighth nerve and the connection between its physiology and pathology and subjective ear sounds naturally lead us to inquire concerning nerve centers which govern

other special senses, and to seek some analogous condition associated with abnormality of these centers in patients presenting such disorders. It is with special reference to these analogies that we present this paper.

Exhaustive studies have been carried on to determine the alterations which take place in the cortical centers after the enucleation of the eyeball, and in connection with such investigations we have questioned patients as to any "sight perception" experienced after their eyes were removed. There are numerous patients with amputated legs who project pain or other sensations in their toes, though this, of course, is not entirely analogous. It does seem, however, that there is a certain so-called "sense of vision" in some of these blinded eyes, though undoubtedly this sense is a visual projection of the imagination or memory, or both. These are manifested by a feeling that some patients have that they "see" a dark grey or even a colored pattern, which they attempt to describe as projected before their eyeless orbits. There are pathological retinal or optic nerve conditions where seeing patients with eyes tightly closed, or in absolute darkness, will claim to see designs or other objects, some of them highly colored, and describe them in general or in detail. I have had a totally blind patient state that he seemed to "see in his field of vision" a boy on a bicycle that was constantly pedaling and yet never moved out of the range of his "visual fields." It at once becomes obvious that it is exceedingly difficult to differentiate visual hallucinations from actual visual pathway irritation and thus we might better give some slight passing consideration to hallucinations. Concerning this, Edmund Parish quotes Schule as follows: "If," he argues, "in cases in which hallucinations occur after blindness and atrophy of the optic nerve of many years' standing (Rudolphi) or with softening of the thalami (Esquirol), we assume that the co-operation, anatomic and physiologic, of the 'sense' is essential, we must suppose that the sensory tract in all its ramifications is involved with the cortical sphere in a pathologic reaction. It is improbable, however, that the whole of the nerve path is implicated, and if we assume an intellectualizing of the perceptions as they ascend, the degree of sensory quality in a hallucination may be taken as a functional

expression of the distance from the periphery of the nerve concerned. The pathologic process may be conceived as a condition of heightened irritability with a specific morbid function; two causes may be assigned for this heightened irritability:

(a) A weakening of the cortical inhibition and consequently increased independence of the sensory centers.

(b) Direct heightening of the irritability, generally resulting from some disturbance of assimilation."

This is, of course, only one of the many theories brought forward in the effort to explain visual and other hallucinations. We certainly would not be justified in classing tinnitus as a hallucination, except in cases of well marked mental disturbance, indicated by symptoms other than the fact that the patient "hears noises." But, is it unreasonable to suppose that in cases of tinnitus with complete deafness we have a certain amount of "intellectualizing," as suggested by Schule? A survey of the literature together with our experience leads to the conviction that the persistence of subjective vision when the optic nerve has been destroyed may possibly be compared to the persistence of tinnitus when we have every reason to believe all possibility of hearing is gone. We feel that there is an analogy between the so-called "seeing stars" and "hearing a big rush or noise" as frequently described by patients subjected to head trauma. We might note here, too, that it is easier to believe drug tinnitus (as associated with quinin and other drugs) affects the center of hearing rather than the nerve tract when we bear in mind that in quinin amblyopia the fundi may appear absolutely normal.

#### TASTE AND SMELL.

Greenwood, in his *Physiology of the Special Senses*, begins his chapter on Taste and Smell, by saying "the senses of taste and smell have many points of resemblance, one being the scantiness of our knowledge respecting the physiology of either." This is certainly not very encouraging. Pursuing our search for an analogy still further, we learn from him that "whether the gustatory cells are really end organs in the sense in which this term may be applied to the retinal cones or rods,



or whether they merely act as props upon which the nerve endings twine, is a matter of doubt. Likewise we have no knowledge as to the cortical representation of taste, hardly even a conjecture worth repeating. There is a tendency to pick out the anterior sylvian convolution as the cortical 'center,' but the evidence is trifling." Recorded cases of perversion of taste and smell are sufficiently numerous, but it is difficult to trace any analogy between them and cases where abnormal sight and hearing are concerned, because we are not sufficiently well informed concerning the relation of the nerve endings and the governing centers. The cortical representation of smell is doubtful; some results suggest the hippocampal region, but the connections of the olfactory tract are numerous and complicated, while experimental work on lower animals is beset with serious difficulties. Some of these animal experiments would almost lead us to believe that smell, at any rate, does not wholly depend upon the olfactory nerves. Greenwood relates a case of Magendie's where the olfactory nerves in a dog were destroyed right down to the lamina cribrosa. The animal recovered from the operation and Magendie presented it with several paper parcels of the same size, some containing cheese and others wood. The dog selected and unwrapped the packets of cheese without hesitation."

#### CUTANEOUS ANALOGY.

Again we have taken up the consideration of cutaneous reactions, attempting to correlate an analogy between tinnitus and pruritis, formication and other forms of cutaneous irritations and reactions. Formication is a term used to describe the feeling of insects, such as ants, persistently running over certain areas of the skin. We had one case of this that persisted for years in spite of every conceivable treatment. It involved the supraorbital branch of the right side. This was in a very active and well-known patient and annoyed him to such an extent that he almost had to give up his business. The symptom was entirely subjective as there was absolutely no indication from the appearance of the skin that there was any pathology. Consequently the evidence, while sufficient to suggest, is not conclusive enough to definitely prove an analogy here again.

Therefore, it seems difficult, though perhaps not wholly impossible, to establish definite analogy between tinnitus and other abnormal physical manifestations. A wide array of evidence has been lined up by neurologists and psychologists, tending to prove that tinnitus is, in a large percentage of cases, of psychic or neurasthenic origin. Woakes brings forward an ingenious argument to prove that Mahomet and Joan of Arc were probably sufferers from tinnitus, induced by the "damp and unhealthful" conditions under which they lived, the prophet in a mountain cave, the Domremy shepherdess exposed to the inclemency of the weather while following her occupation. This might be termed a combination of physiologic and psychic tinnitus. The idea is perhaps amusing, but merely serves to lead us back once more to the subject of illusions. Parish says: "In auditory delusions, the lowest degree is represented by the 'psychic' hallucinations of Baillarger. These are 'soundless' internal voices, which seem to the subject to be addressed to him from outside; they are spoken of by the insane as 'spiritual' or as 'soul language.' By their soundlessness they are clearly distinguished from more highly externalized acoustata, where the 'sound' element is more or less strongly marked, the voices sometimes seeming to whisper softly in the ears, or to be heard faintly from a great distance, and in other cases sounding loud and distinct. Nonvocal hallucinatory noises, such as the ringing of the doorbell, steps in the hall, or in the room itself, knocks at the door, etc., seem as a rule to be indistinguishable in intensity from corresponding objective sounds. Sometimes in dreams, the hallucinatory noises are said to be loud enough to waken the sleeper. In such cases, however, we are often dealing, not with hallucinations, but with external noises heard with abnormal intensity in a state of dissociation. In other cases it may be subjective sensations which are hyperesthetically perceived. For instance, certain attacks (not, of course, to be confounded with epileptic seizures) called by Weir Michell 'sensory shocks,' occur with alarming violence in neurasthenic and hysteric subjects, and after the excessive use of tobacco. On going to bed—not on waking—and while going to sleep a sudden shock is felt like a blow inside the head, in most cases accompanied by a sensation of sight, hearing or smell so intense, that these attacks, often preceded by an aura, are actually

dreaded by those subject to them. This observation seems to confirm Hoppe's view, that "the frequently reported sensation of a loud crash or jar is to be taken as a symptom of fatigue." "4

However, otologists see nearly exclusively cases of tinnitus which are undoubtedly of purely nonpsychopathic origin, and cannot be referred to any psychic cause. Often the subjective sounds are so obtrusive as to disturb the patient's sleep, or cause insomnia. It is probable that the disturbance of sleep may occur because of the absence of outside noises during the night, and also because the patient's mind is not distracted from his affliction by outside occupations, as is the case during the waking hours of the day. The "crash" or "jar" mentioned by Parish as a psychic disturbance is not infrequently encountered in persons of perfectly normal mentality.

A few isolated case reports tend to throw a little light upon this very obscure subject. Such a one was reported by Jones in his manual designed especially for those engaged in the examination of candidates for the air service.

Patient was operated upon by Dr. C. H. Frazier for persistent tinnitus following a fracture of the base of the skull. Dr. Frazier divided the left eighth nerve in the posterior fossa. Shortly after the roaring and ringing almost entirely disappeared. After the patient had enjoyed freedom from this symptom for four months, however, the tinnitus returned. Three years after the operation, the tuning fork test showed complete deafness in the left ear, and douching of this ear failed to produce any of the responses in nystagmus, vertigo, past pointing and falling. Turning to the right, which chiefly stimulates the left ear, caused a very poor nystagmus, vertigo and past pointing, whereas turning to the left, stimulating the right ear, showed fairly good response. This examination indicated that the operation not only completely severed the eighth nerve, but that there had been no regeneration of either its cochlear or vestibular portion. "This case is of interest in that it may throw light upon some phases of the subject of tinnitus. At first glance it would seem to bear out the contention of those who maintain that tinnitus is caused by a lesion within the labyrinth. For four months after the severing of the eighth nerve the patient was comparatively free from noises in the

head. The recurrence of the tinnitus, however, shows without question that in this case at least there occurred after the severing of the eighth nerve, a further degenerative process, central to the point of section, and capable of producing tinnitus. Before operation it would seem reasonable to consider that the process attacked the cells within the spiral ganglia peripheral to the point of section. This would account for the patient's temporary recovery from the tinnitus. The subsequent ear tests showed that the eighth nerve was completely severed, and yet the tinnitus recurred. This would indicate that the tinnitus is most probably produced by a further toxic involvement of the ganglionic cells along the course of the auditory nerve tracts or at their termini in the cerebral cortex."<sup>5</sup> There are other cases recorded where severing the auditory nerve distally did not relieve the tinnitus.

The association of tinnitus with such diseases as "boilermakers' deafness" hardly needs any emphasis here. Many years ago Peter McBride pointed out "there can be no doubt that when the ear is frequently exposed to loud sounds, as in the case of boilermakers, artillerymen, etc., the often repeated concussion of the labyrinth tends at last to cause deafness. Not infrequently the labyrinthine lesion is associated with the middle ear catarrh, which makes the organ of hearing a '*locus minoris resistentiae*.' The loud sound acts primarily by driving the drum membrane inward and with it the stapes." It is obvious that if, owing to eustachian obstruction, the air within the tympanum be rarified, this will be more easily and more fully accomplished, and thus the common association of boilermakers' deafness with tympanic disease is probably accounted for. In these cases the deafness is generally associated with tinnitus, vertigo being uncommon."<sup>6</sup>

In the more than thirty years which have elapsed since McBride wrote, we have not advanced very far in our knowledge regarding the connection between tinnitus and "ear splitting" noises accompanying one's regular vocation. It would be interesting to obtain statistics as to whether the "traffic cop" is subject to any auditory lesion because of his ever recurring shrill whistle, or whether tinnitus occurs more frequently in noisy or quiet locations. We have not been successful in locat-

ing any such reports in the otologic literature. Neither have we been able to locate any injuries, as war injury, to the cortex over the hearing center, which have caused a loss of a previous tinnitus.

Patients complain of high pitched tinnitus much more often than of tones of the lower scale. Could it not possibly be due to the center for high pitched sounds being the last acquired evolutionarily and, therefore, more vulnerable?

While we believe a peripherally caused tinnitus may finally develop into one dependent on central impulse or irritation, and, therefore, continue as such, we have but little evidence to prove it. It does not seem so probable that the central type will become peripheral. Assuming then, that tinnitus is of central, rather than of peripheral origin, much more than generally conceded, we would be greatly pleased to explain a reason for it. This, unfortunately, we cannot do. We could offer the old theory, as threadbare from use as it is difficult to prove, that it was because of the special selectivity of the organisms or their so-called toxins on the highly specialized auditory nerve or center, but we cannot explain this selectivity. It cannot be because hearing is so highly specialized, as other senses are just as elevated in the scale of evolution and perhaps vision is still higher, while the functions of an active cerebrum are, quite probably, much higher than any of them.

There may be a possibility that the causative factor may be a general sclerosis of the nerve or cortex or even an ossification or calcification. As tinnitus is more common in the middle aged and elderly, there would seem to be a possibility of its being a senile change, and that in some cases where it occurs in younger subjects, it is merely evidence of premature senility. While tinnitus may be due to one cause, it most probably has multiple causes eventuating in this one annoying symptom often accompanied with a diminution in hearing.

In spite of our study of the subject of tinnitus, its causes remain just as obscure as it was before we directed our humble efforts towards its solution. We can but conclude that, while it may be due to one cause, it most probably has multiple causes, eventuating in this one annoying symptom which is so often accompanied by diminished hearing.

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LXXXVIII.

RADIOTRANSLUCENT FOREIGN MATERIAL IN  
THE LARYNGO-TRACHEO-BRONCHIAL TREE.\*

By EDWIN MCGINNIS, A. B., M. D.,

CHICAGO.

The word radiotranslucent I am using in the sense that the light rays of the X-ray machine pass through the objects without sensitizing the plate—that is, without casting the shadow that would make the obstructing object visible in an X-ray picture. Pennies, telephone slugs and other metal objects are, of course, not radiotranslucent. Their presence in the bronchial tree is readily registered on the X-ray plate. But other objects, many of them vegetable in nature, may be said to be radiotranslucent, in that the X-ray light rays pass through them and so leave their presence undiscovered.

Differential diagnosis is sometimes difficult. Therefore, the experience of the physician is a great determining factor. Some of the mistaken diagnoses that come to mind are: spasmodic laryngitis, laryngeal diphtheria, edema of the larynx, asthma, asthmatic bronchitis, acute and chronic bronchitis, and even pneumonia.

A number of cases of radiotranslucent objects, such as bits of eggshell caught in the larynx between and parallel to the vocal cords, have been observed. Also cases with thin pieces of chicken scapula. Small thin flat buttons have also been the offending objects. These produce very little obstruction to breathing, but in babies the crying voice is altered; and in one case the youngster seemed unable to make an audible cry.

Sometimes these radiotranslucent objects are sharp fish bones stuck into the laryngeal mucosa; in other cases the object slips through the laryngeal chink and gets into the trachea. In this situation, if the object is large, obstruction symptoms are present, difficulty in respiration and cyanosis. Nuts, prune pits, kernels of corn, beans, peas, glass and other types of

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\*Read before the Chicago Laryngological and Otological Society, March, 1927.

beads, and even particles of food are liable to be in situ. An aid in the diagnosis in this situation is the noise of the object sliding up and down the trachea. Jackson has called attention to this.

Tracheal location does not produce much variation in physical findings in the chest unless the object has been pulled more into one main bronchus than into the other. Mistakes in diagnosis have been made, even in the best regulated of hospitals, as evidenced by the following case: Youngster had had access to some pistachio nuts and one of these evidently slipped into the trachea. The accident was forgotten or not noted by the parents. Child was admitted to the hospital for observation and was around about a week. Coughing spells and occasional respiratory crises were present. Nothing much was thought of this, until one afternoon the child had a sudden permanent respiratory obstruction and a crisis was at hand. Tracheotomy was performed without relief to the breathing, and the child died. Postmortem revealed a pistachio nut wedged into the bifurcation so tightly as to completely obstruct air flow. This was an emergency where experience in this work would have helped.

When round objects are inspired which are small enough to slide into one main bronchus or the other, they usually come to rest in the lowest possible point. In this case physical findings and symptoms will be one sided. If the object is of a nature to swell, it will ultimately shut off the entrance of air into the main bronchus. The breath sounds will be absent, and if the object remains long enough the lung will collapse. I have seen cases of lung collapse which were also due to an excessive amount of thick mucus caused by the presence of peanut particles in the bronchi. There have been other cases in which I am sure that the thick, gelatinous like content of a chronically infected antrum of Highmore was inspired during an operative anesthetic and obstructed a main bronchus.

Blood clot following operations on the nose and throat could also be inspired and close off part of the bronchial tree. I have seen two such cases.

If the inspired object just about fits into a terminal lobe bronchus, it will shut off the air entering a lobe. This is common in the lower lobe bronchi.



I have had it proved often that a careful history is important because in giving it the statement of the accident usually comes out. The individual has usually been perfectly well up to a certain time. Chest X-ray, as we have seen, cannot in cases of radiotranslucent objects, give much information, and one should not, therefore, be misled by the absence of X-ray evidence. Diagnostic bronchoscopy, however, usually aids in the diagnosis and cure of the individual.

A few recent illustrative cases will bring out vividly some of the points. A youngster, about four years old, had been seized with a sudden choking spell while feeding the chickens corn. She ran to her mother in her difficulty and was rather cyanosed at the time. Cough and cyanosis subsided, but within twenty-four hours spasmodic cough commenced. Cough became productive, and during a period of three weeks several physicians examined her and X-ray pictures were taken. At last the possibility of something in the bronchus was thought of and she was sent in for examination. The prominent thing in the history was that the mother said "she was perfectly well until that day, and she was shelling an ear of corn to feed to the chickens. Since then she has been sick with a cough and has lost a good deal of weight." Physical examination of the chest revealed moist râles limited to the right chest. Bronchoscopic examination revealed a white Dent kernel of corn in the right main bronchus. This was removed with forceps and child made a good operative recovery. Letter two months subsequent from child's mother stated that her daughter made a prompt, permanent recovery and regained all her lost weight.

Boy, about three years old, while eating a luncheon consisting of a piece of bread, one pickle and a small amount of meat, suddenly strangled, and after a severe fit of coughing seemed to be relieved of his tracheal obstruction. The next day an occasional cough developed. Pediatrician was called in, who suggested X-ray examination. This was deemed negative. Youngster was under observation for about two weeks, as he still had his chest upset. Consultation with a laryngologist who had had a good deal of experience resulted in the diagnosis of foreign body in the right main bronchus. He was sent in to me for bronchoscopic observation. The morning he landed in Chicago he broke out with measles rash and we

had to send him to a contagious hospital. I saw him about every day until his convalescence, and one day I discovered that the attending physician was looking upon this boy's chest condition as a measles bronchitis. About the time of dismissal from the hospital he sprang a rather high temperature and a foul smelling productive cough. An immediate bronchoscopic examination revealed a small hard object in the right bronchus. On removal this proved to be a piece of bone. Aspiration of a large amount of foul mucopurulent secretion was done at the same time. One dose of neoarsphenamin cleared up the odor, and the lad made a rather rapid recovery.

Another case of aspiration of a kernel of corn was first treated as a laryngeal diphtheria with a rather large dose of antitoxin. The youngster had a rather severe serum reaction with quite a little laryngeal edema. Removal of the offending object helped some in the relief of the youngster's breathing. Laryngeal and tracheal swelling became more pronounced, and the house surgeon, who had had a good contagious service, intubated and carried the youngster along for thirty-six hours; then he extubated, but the youngster could not carry on without the tube. I did a low tracheotomy with marked relief.

In conclusion, adults, young children, and infants especially, are prone to put things into their mouths. In the vast majority of cases this has no ill effects, but occasionally the blast of inspired air carries the object into the air passages. The accident with its accompanying symptoms of cough, difficulty in breathing, sometimes transient cyanosis are noted by the individual and attendants, and should never be passed over lightly by the attending physician.

The individuals have usually been perfectly well before the accident, but afterwards there are present certain prominent symptoms resulting from the ultimate resting place of the inspired object.

104 SOUTH MICHIGAN AVE.

ACUTE NONSUPPURATIVE OTITIS MEDIA AND  
SEQUELÆ: A REVIEW OF ONE HUNDRED  
CASES.

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By far too many instances of so-called conductive deafness are the sequelæ of untreated acute nonsuppurative otitis media. These nonsuppurative cases seldom consult an aurist until the end result of their condition, namely, deafness, is marked enough to be troublesome. It is useless to blame the patient, for there generally is little if any pain connected with this condition, but unfortunately too many general practitioners regard earache, its presence and degree, as a criterion on which to base prognosis and the necessity of consulting a specialist. Until earache is forgotten and stuffiness and tinnitus taken as the danger signals, we will all of us have cases of deafness which might have been prevented.

The acute nonsuppurative inflammations of the middle ear may be divided into two large groups: (1) Those cases in which no fluid can be demonstrated in the middle ear; (2) those cases in which it is possible to demonstrate fluid in the middle ear. Both groups have many signs and symptoms in common, and it is essential that as minute a diagnosis as possible be made, in order that proper treatment may be instituted at once to prevent deafness. Even the experienced aurist will sometimes find it difficult to tell just what condition he is dealing with. When there is no exudate, it is sometimes difficult to decide whether the condition is acute or whether a chronic condition has advanced enough to suddenly give symptoms. Where there is an exudate, even greater difficulties are met with. It is a very inappropriate time to discover an underlying chronic lesion when a patient should have recovered from an acute one, for then the discovery is of little benefit to anyone.

Let us first consider those cases in which no fluid can be demonstrated. Textbooks refer to acute salpingitis as a very usual accompaniment of acute rhinitis, which subsides as the rhinitis is treated. While this is true in many cases, in many more it does not hold good. Much oftener the acute rhinitis is an accompaniment of an acute flareup of a chronic salpingitis which is caused by some pathologic lesion close at hand—a most important distinction and one that should always be made.

If the salpingitis is merely the accompaniment of an acute rhinitis, the tympanic membrane, whether congested or not, is always of normal thickness, neither hypertrophied nor atrophied. If the membrane is found either hypertrophied or atrophied, immediately look further for a cause. The acute rhinitis may be regarded as of slight importance.

Consider first a case in which the tympanic membrane is hypertrophied. These patients give a history, that with a cold their ears are sometimes stuffed up but not always.

There are two chief reasons for this hypertrophy. The first is intermittent pressure. The usual cause of intermittent pressure is chronic infected tonsils. They are often small and adherent, while the lymphoid tissue behind the posterior pillars and at the orifice of the eustachian tubes is hypertrophied. So seldom does a pathologic nasal condition seem to cause this intermittent pressure, that where there is hypertrophy, even though the tonsils appear normal and the nose pathologic, it is well to look at the tonsils again. Their removal will often give a good result, even if a nose that is not perfectly normal is left alone.

The other great cause of this hypertrophy is chronic inflammation of the drum and surrounding tissues of the middle ear. Both these factors are generally present, and if we could tell which was the greater factor in any case under consideration our prognosis could be more freely given, as well as our mode and length of treatment.

Unfortunately we have as yet no definite criteria to decide this, but the history together with the physical findings should aid us a great deal. More particularly we should note the length of time the patient has noticed any deafness, the degree

and type of deafness as we find it. The amount of hypertrophy—is it slight or marked? Is the drum retracted, for if it is, adhesions are generally causing the greater part of the retraction, for intermittent pressure alone does not seem to cause much of it.

*Cases of More Than a Year's Duration of Slight Deafness, Which Have a Hypertrophied Drum.*—With a slight hypertrophy, there is generally in these cases some reaction of the drum, which would indicate there were probably some adhesions from a previous mucous exudate, the cause of which may well have disappeared. If an active lesion is found anywhere near, remove it for preventive reasons, because that patient is going to get worse unless everything possible is done. Do not allow this type to react too much, as even long continued inflation may at the best only arrest the condition.

With a very marked hypertrophy in this type of case, there is seldom much if any retraction. These cases seem to be purely mechanical, and excellent results are generally obtained by radical removal of the cause, usually the tonsils. These cases require but little after treatment.

*Cases of More Than a Year's Duration of Pronounced Deafness Which Have a Hypertrophied Drum.*—If the hypertrophy is slight, the retraction is always marked and the pathologic lesion is essentially inflammatory, and adhesions are causing the deafness. Operative or any other form of treatment does little or no good, and only serves to discredit treatment of any form of deafness. I have seen these cases inflated for years, but never saw any good from it. The original lesion may have been nasal, certainly not tonsillar. But the essential lesion is the result of an untreated mucous exudate in the middle ear.

If, on the other hand, the hypertrophy is marked, often there is not much retraction, and then something may be done. If the tonsils are infected, remove them and also use inflation. This often greatly benefits the patient, though we can demonstrate no improvement. The process has stopped instead of getting slowly worse, and the patient seems to get along much better. I have not had a case in which the removal of any nasal condition seemed to give any improvement.

*Recent Cases of Slight Loss of Hearing Which Have a Hypertrophied Drum.*—In this type of case, the greater the amount of hypertrophy the graver the prognosis. Any great amount of hypertrophy must be inflammatory; therefore, the danger of adhesions is much in proportion to the amount of hypertrophy. Remove the tonsils, correct any other pathologic lesion and use inflation, according to the degree of hypertrophy. Results are generally good.

*Recent Cases of Marked Loss of Hearing, Which Have a Hypertrophied Drum.*—In this type of case, it is the amount of retraction rather than the degree of hypertrophy that indicates to us how much benefit is going to be derived from treatment. If the retraction is slight, the case probably has had an untreated mucous exudate and will continue to get worse. All pathologic lesions should be cleared up and inflation instituted at once and kept up for a long period, but the patient should be warned that he may get worse in spite of our treatment, but not nearly as rapidly as though nothing were done.

If, however, the retraction is great it may only be mechanical, and often great benefit is derived from the removal of the offending cause, the tonsils, and a short period of inflation.

*Cases With Atrophy of the Drum.*—Here an altogether different problem confronts the aurist. As a rule, the patients say that with the slightest cold their ears are stuffed up, but not as markedly so as with the hypertrophic type; the constancy is the feature here. There is continually more or less blocking of the eustachian tube, and this continuous pressure causes the atrophy of the drum.

Adhesive processes are unusual in these cases, for that would mean inflammation with its consequent hypertrophy. The amount of atrophy, the degree of retraction, the length of time they have suffered, and the degree of deafness go hand in hand.

For the cause of this condition we look in the nose, for it is generally some pathologic lesion there that is causing the trouble. It is rare for the tonsils, whether diseased or not, to have anything to do with it. Considering these facts, the correction of the nasal condition with inflation and painting the eustachian tube with some astringent generally stops

the process, and may even improve conditions somewhat. This is a great relief to the patients, even though we can detect no improvement.

To sum up, as a general rule, a hypertrophic drum means a tonsillar cause while an atrophic drum means a nasal cause.

To proceed to the cases of Group 2, where there is fluid in the middle ear. Again our symptoms will be practically the same, namely, slight deafness, a feeling of stuffiness, sometimes tinnitus and sometimes slight pain. We find a negative Rinne, bone conduction increased, low tones (C64) not heard, etc. These cases require a very careful investigation. What will be our prognosis and what is the best treatment to give them? Shall we do a paracentesis of the drum as well as use inflation?

In regard to otoscopic examination of the drum, the conditions of hypertrophy or atrophy, though sometimes very difficult to determine, should be very carefully noted as they may have the same significance as in acute salpingitis.

At the time, though, the most important matter to be determined is whether the fluid, if allowed to remain, will later cause adhesions. If adhesions are going to form because of its remaining there, one must do a paracentesis and remove the fluid by inflation or suction. The tendency of the fluid to form adhesions depends on whether it is a transudate or an exudate. In other words, is the fluid serous or mucous? Which it is cannot always be determined for a certainty. Then regard it as a mucous exudate and do a paracentesis. This is not a serious operation and one is on the safe side.

With a serous transudate the drum is unchanged from its usual structure, and although bulging, it never loses its luster. If tinnitus is present, which is not a constant symptom, both it and the deafness are improved by inflation. If either the tinnitus or deafness fail to improve from inflation, a paracentesis should be done followed by inflation.

With a mucous exudate tinnitus is always present, and neither it nor the deafness is improved by inflation. As the process is inflammatory, the drum always shows some change ranging from a slight congestion with a little loss of luster to a thick, gray, lusterless membrane. These cases are followed by adhesions which later give adhesive deaf-

ness. A paracentesis should be done and a bit of dry cotton placed next the drum.

On inflation, if the case is at all recent, you will recover mucus on the cotton. Microscopically it will be found to contain mucous cells and some leucocytes. If the case is of seven or eight days' duration, you may get very little mucus, but you will always get some. If one does not get any moisture at all on the cotton and yet the findings have indicated the presence of a mucous exudate, one is, in all probability, confronted with an altogether different pathologic condition, namely, a streptococcus infection.

This specific infection is often practically painless and generally involves the mastoid antrum and the middle ear simultaneously, and a very guarded prognosis must be given, as mastoiditis is apt to occur.

The opening in the drum should be kept patent and inflation performed each day until the ear is dry and the tinnitus ceases.

YARMOUTH CLINIC INFIRMARY.



## Abstracts of Current Articles

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### **On the Occurrence of Brain Tissue Within the Nose: the So-Called Nasal Glioma.**

*Douglas Guthrie, M. D., and Norman Dott, M. D., Edinburgh.*

Guthrie and Dott feel "that the occurrence of brain tissue in the nasal cavity as a development from an embryonic rest of the central nervous system is sufficiently common in young children to constitute a definite clinical entity." These authors stress the intimate relation between the frontal lobes of the brain and the upper part of the nose in the region of the cribriform plate, and also the fact that the cribriform plate may vary considerably in structure, but rarely reveals congenital dehiscences. Malignant nasal growths which extend into the brain occur occasionally, but it would seem that growths from the brain are rarely incorporated into the nasal cavity. Hernia of the brain may occur at the site of a fracture in the anterior fossa.

These authors also give a very plausible explanation of the frontal and intranasal encephalocele occasionally found in infants as the result of faulty development during embryonic life. A small bud of brain tissue grows out from the embryonic brain and as the skull develops this "bud" may become completely separated from the brain proper or may remain connected only by a pedicle. Although cases of congenital tumors are frequently reported under the term "intranasal glioma," this classification is hardly correct, as the glial cells which are found within the tumors are fully developed and show no embryonic character.

Two cases are reported in detail:

Case 1.—The patient was a man, 33 years of age, who presented no history of nasal or aural disease. First symptoms were headache behind eyes and in left temporal and suboccipital region, some nausea, drowsiness (later), bilateral choked disc, marked drop in pulse, subnormal temperature. A left subtemporal decompression was performed. The brain was found to be very tense. A gliomatous mass was suspected but no at-

tempt was made to remove it. The patient died on the 13th day after the operation. A very meticulous postmortem examination was made. It was found that a tumor projected through the cribriform plate into the left ethmoid region of the nasal cavity. Pathologically this case was diagnosed as neuroglioma. Complete sections were made through the gliomatous protrusion which had penetrated into the nose.

Case 2.—The patient had been in good health up to the age of 46 years when he was struck by a beam and rendered temporarily unconscious. The accident was accompanied by profuse bleeding from the nose. A gradual paralysis of the right arm and leg appeared, and persisted throughout the following year. Although the patient had frequent headaches, he never suffered from optic neuritis, but he did experience several mental changes. The above symptoms went on for nine years, at the end of which time he was found in an unconscious condition and remained in that state for five days, following which he regained his former mental state.

On two occasions during the following year growths which seemed to be ordinary nasal polypi were removed from the left nasal cavity; the tissue of the growths, however, was not examined microscopically. After a period of five years, the nasal obstruction reappeared and the patient came under the care of Dr. Guthrie, who on examination found a soft grayish tumor which appeared to be a nasal polypus. When this growth was being removed it burst and discharged a straw colored fluid. The exact site of origin of the tumor was not determined, but satisfactory relief from nasal obstruction was gained for the patient by its removal—otherwise the patient's condition remained unchanged. A section through the entire mass, when examined microscopically, showed the underlying characteristic features of a nasal polypus, with the addition of glial cells which were fully formed but had none of the embryonic characteristics which are suggestive of a true glioma. The authors believe that the tumor within the nose bore no relation to the intracranial lesion. They think that the best descriptive term for this tumor is encephalocele, since the glial tissue had been present in the nose since birth and had later become incorporated in the nasal polypus.

Two valuable suggestions are made by the authors: (1) That a microscopic investigation of all nasal polypi might reveal the presence of neuronc tissue in a certain proportion of the cases; (2) that investigation of the olfactory function in the first case cited would have been of great value in diagnosis.

A complete review of the literature is appended. M.

#### **Endocrine-Pituitary Correlations.**

L. Maffeo (Naples), *Arch. Ital. Otol.*, 38:172, August, 1927.

Based on histologic research on the nasal mucosa of the dog, Maffeo claims that pregnancy causes diminution of mucosal secretion and hyperplasia of connective tissue. Influence of the ovarian hormone is diverted from its general activity to the special problem of reproduction at such a time. He suggests that long standing endocrine disturbances in the sexual sphere may readily lead to atrophic mucosal changes. In further experiments, he is attempting to explain the mechanism of vicarious menstruation and related upper respiratory phenomena.

F.

#### **Carcinoma of the Larynx With Myotic Invasion.**

R. Motta (Rome), *Arch. Ital. Otol.*, 38:515, August, 1927.

In two cases of carcinoma of the vocal cord with mulberry-like pale red granulations, not ulcerated, sections showed a proliferating mycelium between the cells, and on culture, the mold *Hormodendron Cladosporoides* was identified.

F.

#### **Influence of the Cervical Sympathetic on Nystagmus.**

U. L. Torrini (Florence), *Arch. Ital. Otol.*, 38:734, December, 1927.

In spite of marked congestion of the external ear in rabbits in which cervical sympathectomy had been done, no apparent difference in nystagmic reactions could be elicited as between the sound and the sympathectomized ear.

F.

#### **Lesions of the Cervical Sympathetic Ganglia and of the Carotid Plexus.**

E. Lasagna (Parma), *Arch. Ital. Otol.*, 39:1, January, 1928.

Prof. Lasagna declares that explanations by various authors of the temporary results of periarterial sympathectomy are purely imaginary. He reports results of numerous animal experiments. Removal of the entire chain of sympathetic ganglia provokes congestive changes in the upper respiratory tract,

which rarely last more than a month, with restitution of the original conditions after that time. Duration of these changes is increased by simultaneous pericarotid sympathectomy; this procedure alone is as effective as ganglionic removal. Nevertheless, many fibers remain untouched by either process. F.

**Ethmo-frontal-sphenoidal Mucocoele Operated Intranasally.**

*E. V. Segura and H. Zubizarrieta (Buenos Aires), Rev. Oto-neuro-oftal., 1:329, December, 1927.*

An excessively large mucocoele of ethmoidal origin, invading the frontal and sphenoid cavities, caused little pain in spite of great exophthalmos. Segura removed the middle turbinate, taking out the mesial wall of the huge cavity. Exophthalmos gone in two months, and perfect health past three years. He recommends the intranasal route wherever possible. Rejecting theories based on sympathetic dysfunction, Segura inclines to the retention cyst idea, with possible congenital maldevelopment as the exciting factor. F.

**Origin of Forced Movements, in Relation to Labyrinthine Function.**

*K. Ono (Tokyo), Jap. J. Med. Sci., XII, Otorhinolar, 1:1, Sept., 1927.*

Phenomena due to destruction as well as to irritation of the vestibular apparatus must be interpreted as irritative. Ono considers both increase and decrease of excitation of the vestibular apparatus as but a signal to the cortical centers. Peripheral organs of posture (muscles, etc.) receive these stimuli only indirectly, through such central excitation. Forced movements, even the "rolling" movements of Flourens, are but the expression of an effort to adjust the body to an apparently altered center of gravity. Nystagmus represents a similar effort of the eyes to adjust themselves to such apparent alterations. Ono publishes the results of years of animal experimentation, but his photomicrographs were lost in the Tokyo earthquake. This is a most exhaustive monograph, occupying 72 pages of this excellent new publication. F.

**Experimental Meningeal Labyrinthitis.**

*Y. Honda (Tokyo), Jap. J. Med. Sci., XII, Otorhinolar, 1:73, September, 1927.*

By injection of various organisms into the spinal canal, Honda has demonstrated that infection enters first by the aque-

ductus cochleæ, then the scala tympani, thence the middle ear. Final stages include invasion of the scala vestibuli and the aqueductus vestibuli, with the semicircular canals and their contents. Such circumscribed invasion of the scala tympani without vestibular involvement corresponds to the usual situation in deaf and dumb individuals. F.

**Basal Phlegmon of the Tongue.**

*A. Viela (Toulouse), O., R., L., Internat., 12:49, February, 1928.*

Anatomic study convinces Viela that these abscesses develop from broken down lymph glands in the space between the genioglossus and hyoglossus, along the course of the lingual artery. To avoid extension into deeper structures, these abscesses should be opened either above the hyoid, or better, by a trocar inserted at the side of the tongue, directed backward, down and medialward. Dilation with a grooved director or forceps will suffice for complete relief. F.

**Thyroglossal Cysts and Allied Conditions.**

*N. Patterson, J. Lar. and Otol., 43:313, May, 1928.*

This exhaustive review directs attention to the central location of the thyroglossal duct and to its passage in front of the hyoid bone. It should be distinguished from suppurating glands, dermoids, lipomas, angiomas and from tubercle or syphilis of the larynx. Injection of radio-opaque fluids is recommended, and prior to surgery, gentian-violet or methylene blue may be used for identification. Sistrunk's total excision by the supra-hyoid route is recommended. It should be remembered that aberrant thyroids may occur anywhere along this duct. F.

**Otologic Aspects of Peripheral Facial Palsy.**

*P. Bertein (Paris), O., R., L., Internat., 12:5, January, 1928.*

Rejecting theories of chilling as causative of peripheral facial palsies, Bertein evokes the notion of reflex paralysis caused by a transitory congestion of the facial nerve, due to stimulation in the field of the trigemina. He cites two cases of Barré and Leriche in which transitory facial palsy followed intracranial section of the trigeminus. Redness of the tympanic membrane he considers a sign of reflex vasodilatation, not inflammatory. F.

**Influence of Nasopharyngeal and Sphenoidal Disease on the Hypophysis.**

*S. Citelli (Catania), Arch. Int. Lar., 7:129, February, 1928.*

Citelli has, since 1911, ascribed his syndrome (of loss of memory, sleepiness, inability to fix the attention and distaste for work) in children over ten, with adenoids, to an actual anatomic and circulatory relation between the adenoid mass and a persistent though small pharyngeal extension of the hypophysis. This syndrome he finds in about 15 per cent of adenoid cases over ten years. Infantilism, or even the adiposogenital dystrophy of Frölich, may be present and require anterior pituitary therapy in addition to adenoid removal. Closed sphenoidal empyemata and neoplasms of the sphenoid are reported as causing Citelli's syndrome, even in adults of 30 or more.

F.

**Rapid Death of Infants Operated for Mastoid Antrum Disease.**

*G. Canuyt (Strasbourg), Arch. Int. Lar., 7:276, March, 1928.*

Canuyt reviews a great many postoperative deaths of babies operated upon for mastoid antrum suppurations, in shock and with high fever, twelve to forty-eight hours after an apparently successful procedure. Dismissing thymic death, hemorrhage, special types of infection, shock and overwhelming septicemia, Canuyt frankly admits that chloroform anesthesia was at fault. Since he has been using ethyl chloride locally, or no anesthesia, he has had no deaths. His present technic includes myringotomy and a straight, swift postaural incision. He has had no fistulae and no recurrence since adoption of this simplified procedure in 1924.

F.

**Sensory and Trophic Disease of the Pharynx Associated With Arthritis Deformans of the Cervical Spine.**

*J. Terracol (Strasbourg), Arch. Int. Lar., 6:1025, November, 1927.*

In examination of a considerable series of patients complaining of "burning tongue," glossodynia, cervical constriction, foreign body sensation, it was found that no local signs were present in spite of the severest subjective complaint. Radiographs of these paresthetic individuals demonstrated characteristic lesions of the bodies of the middle cervical vertebræ—notably a lipping of the lower anterior rim of one vertebral

body over the next, and a flattening of the body itself. Lime deposits in the prevertebral fascia are frequent.

Considering the factor of pressure upon emerging spinal roots at the intervertebral foramina insufficient, Terracol suggests a chronic vasomotor disturbance, possibly based upon disturbances of the cervical sympathetic. Treatment by diathermy, after epidural injection of lipiodol along the vertebral column, is highly recommended by the author, but results are still doubtful.

#### **Radiologic Examination of Ozena.**

G. Ferreri (Rome) and L. Parola (Milan), *Arch. Int. Lar.*, 6:769, July, 1927.

After discussion of various theories of the pathogenesis of ozena, Ferreri discusses findings in 100 ozenal cases (mainly 15 to 35 years), radiographed by Parola of Milan. Syphilitics and tuberculous were excluded. Aside from the large fossæ in all, and atrophic turbinals in 33 cases, the most important findings were: Arrest of development of the frontals (36 per cent) and rarefaction of the ethmoid (46 per cent), with occasional thinning of the cell walls. Only a few cases of dark antra or thickened sinus mucosa were observed, and these were found due to secondary pyogenic infection rather than to the Perez or Lowenberg bacilli. Ferreri opposes the theory of accessory sinus infection as responsible for ozena, considering more probable the idea of a dystrophy affecting the developing mucosa and budding sinuses through heredity or at an early age.

F.

#### **Clinical Value of Vestibular Nystagmus.**

L. Baldenweck (Paris), *Arch. Int. Lar.*, 6:897, September, 1927.

This long survey brings the work of Jones and Eagleton into critical relation with that of European observers. The author considers spontaneous vestibular nystagmus most important in differential diagnosis. Past-pointing and falling are toward the slow component in peripheral affections; if not, one must suppose an extralabyrinthine lesion, notably of the posterior cerebral fossa. Nystagmus provoked by sudden movement of the head appears to come from the semicircular canals; by slow movements, from the otolith system.

Preferring the minimal stimuli of Kobrak for the caloric test, Baldenweck recognizes the inadequacy of the old theory of convection currents, and points out that the duration of nystagmus depends very largely upon the condition of the central labyrinthine connections. The period of latency, on the other hand, depends upon local conditions and upon the condition of the peripheral labyrinth. Galvanic tests are inaccurate, in his judgment. In addition to the cases of complete loss, Baldenweck draws attention to partial affections, either attacking the otolith mechanism alone, or a single canal. The latter may be worked out by caloric and rotatory tests in various postures; the former, when vertigo and nystagmus occur, in certain head positions. Also dissociated or paradoxical findings may come up in labyrinthine inflammations and in hereditary or acquired syphilis. F.

#### **Experimental Ozena.**

*J. Rebattu and H. Proby (Lyons), Arch. Int. Lar., 6:804, July, 1927.*

Citing various recent experiments on the sphenopalatine ganglion and nerves, the authors report a case of unilateral ozena following retention of a shell fragment in the pterygomaxillary fossa for eighteen months. Damage to the sympathetic fibers controlling nasal circulation is held responsible for the atrophy, but secondary bacterial invasion has altered the mucosa. The case has been studied since injury in 1916. F.

#### **Results of Periarterial Sympathectomy for Ozena.**

*E. Casteran (Buenos-Aires), Arch. Int. Lar., 6:810, July, 1927.*

Reporting two failures, Casteran considers pericarotid sympathectomy useless, because some fibers remain; also regeneration of fibers across the gap takes place within a month. Immediate results are good but transitory. F.



## Books Received

### **Bronchoscopy and Esophagoscopy.**

*By Chevalier Jackson, M. D., Sc. D., LL.D., F. A. C. S., Professor of Bronchoscopy and Esophagoscopy, Graduate School of Medicine, University of Pennsylvania. Cloth. 12mo of 457 pages, with 179 illustrations with ten color plates. Philadelphia, W. B. Saunders Company, Second Edition, 1927.*

This second edition of Dr. Jackson's familiar text is devoted chiefly to the practical and technical problems of endoscopy. In his own words, "this book, intended for use as a working manual, is stripped to the bare essentials." "Canned experience," which he has dubbed his table of references, characterizes the entire work. It is written in words of few syllables and abounds in the usual clear illustrations.

### **Diseases of the Nose, Throat and Ear.**

*By A. Logan Turner, M. D., LL.D., F. R. C. S. E., Consulting Surgeon, Ear and Throat Department, Royal Infirmary, Edinburgh (with the collaboration of J. S. Fraser, M. D., F. R. C. S. E., W. T. Gardner, M. C., M. B., F. R. C. S. E., J. Lithgow, M. B., F. R. C. S. E., G. Ewart Martin, M. B., F. R. C. S. E., and Douglas Guthrie, M. D., F. R. C. S. E.). Cloth. 12mo of 440 pages with 234 illustrations. New York: William Wood and Company, Second Edition, 1927.*

An unusually concise text, characterized by a greater consideration of pathology than usually finds its way into a work of this scope. Many photomicrographs and color plates well reproduced.

### **L'Anesthésie Loco-Regionale en Oto-Rhino-Laryngologie et en Chirurgie-Cervico-Faciale.**

*By Georges Portmann, Professeur de Clinique, Oto-Rhino-Laryngologie a l'Universite de Bordeaux et Paul Leduc, Anesthésiste a la Clinique Oto-Rhino-Laryngologique de l'Universite de Bordeaux. Paper, octavo, 321 pages with 84 illustrations. Paris, Gaston Doin & Cie, 1928. fr. 50.*

A comprehensive work, profusely illustrated. Solutions and methods of injection are briefly discussed, but the major portion of the book deals in detail with the encountering of nerve trunks. Divisions of the work deal with the ears, the nasal chambers, sinuses, larynx, trachea, mouth, jaw, face and neck.

**Technique Chirurgicale Oto-Rhino-Laryngologique.**

By *E. J. Mourc*, Professeur Honoraire a la Faculte de Medecine de Bordeaux, etc., *G. Liebault*, Oto-Rhino-Laryngologiste de L'Hospital de la Glaciere, and *G. Canuyt*, Professeur de Clinique Oto-Rhino-Laryngologique a la Faculte de Medecine de Strasbourg. 8vo of 234 pages with 156 illustrations. Paris: Gaston Doin & Cie., 1928.

**Practical Guide to Diseases of the Throat, Nose and Ear.**

By *William Lamb, M. D., C. M. Edin., M. R. C. P.*, Consulting Surgeon, Birmingham Ear and Throat Hospital. Revised by *Frederick W. Sydenham, M. D., C. M. Edin., F. R. C. S. Edin., D. P. H. Vict.* 16mo of 451 pages, illustrated. New York: William Wood and Company, Fifth Edition, 1927.

**Ohrenheilkunde für den Praktischen Arzt.**

By *Dr. Rudolf Leidler*. Paper. 16mo of 297 pages with 45 illustrations. Vienna: Urban & Schwarzenberg, Second Edition, 1928.

**Handbook of Diseases of the Ear.**

By *Richard Lake, F. R. C. S.*, Consulting Surgeon, Royal Ear Hospital, and *E. A. Peters, M. D., F. R. C. S.*, Surgeon, Royal Ear Hospital. Cloth. 16mo of 310 pages with 80 illustrations. New York: William Wood and Company, Fifth Edition, 1927.

**An Experimental Study of Affects and Associations Due to Certain Odors.**

By *J. H. Kenneth, M. A., Ph. D.* One of a series of Psychological Monographs. Paper. Pp. 64. Albany: Psychological Review Publications, 1927.

**La Syphilis Auriculaire, Oreille et Nerf Acoustique.**

By *J. Ramadier*, Oto-Rhino-Laryngologiste des Hopitaux de Paris. Paper, 174 pages. Paris: Gaston Doin & Cie., 1928.

**The Nose, Throat and Ear.**

By *John F. Barnhill, M. D., F. A. C. S.*, Professor of Surgery of the Head and Neck, Indiana University School of Medicine. Cloth. 8vo of 604 pages with 452 illustrations and four color plates. New York, D. Appleton and Company, 1928.

Another textbook. "This work has been written chiefly for students and practitioners of medicine. The thought constantly has been to present essential facts in brief form. In carrying out this plan, theoretical and controversial discussions are omitted and practices not well established are given little space." A safe and useful book for undergraduates.

**Diseases of the Larynx.**

By *Harold Barwell, M. B., F. R. C. S.* Cloth. 12mo of 278 pages, with 112 illustrations. London: Oxford University Press, Third Edition, 1928.

**Plastic Surgery of the Orbit.**

By *J. Eastman Sheehan, M. D., F. A. C. S.*, Professor of Plastic Surgery, New York Post-Graduate Medical School and Hospital, with a Preface by Pierre Sebileau, Professeur de la Faculté de Médecine de Paris. Cloth. 8vo of 348 pages, illustrated. New York: The MacMillan Company, 1927.

**The Eye.**

By *C. W. Rutherford, M. D., F. A. C. S.*, Associate in Ophthalmology, Indiana University School of Medicine. Cloth. 8vo of 404 pages with 305 illustrations and 12 color plates. New York: D. Appleton and Company, 1928.

**Manual of the Diseases of the Eye.**

By *Charles H. May, M. D.*, Director and Visiting Surgeon, Eye Service, Bellevue Hospital, New York, 1916 to 1926. Cloth. Price, \$4.00. 12mo of 445 pages with 374 illustrations and 73 color plates. New York: William Wood and Company, Twelfth Edition, 1927.

**Handbook of Ophthalmology.**

By *Humphrey Neame, F. R. C. S.*, Ophthalmic Surgeon, University College Hospital, London, and *F. A. Williamson-Noble, F. R. C. S.*, Assistant Ophthalmic Surgeon, St. Mary's Hospital. Cloth. 12mo of 312 pages with 41 colored illustrations and 194 text figures. New York: William Wood & Company, 1927.

**Diseases of the Nose, Throat and Ear.**

By *W. S. Syme, M. D., F. R. S. E., etc.*, Lecturer on Diseases of the Throat and Nose, Glasgow University. Cloth. 16mo of 395 pages, illustrated. New York: William Wood & Company, Second Edition, 1927.

## Society Proceedings

### CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

*Regular Meeting, December 5, 1927.*

THE PRESIDENT, DR. GEORGE F. FISKE, IN THE CHAIR.

#### **Presentation of Clinical Material.**

DR. NORVAL H. PIERCE presented a deafmute child, who had practically no response to the static apparatus. Five blood Wassermann tests had been negative. There was no history of consanguinity and no spinal meningitis. Both branches of the eighth nerve were affected. In the presence of such a history he had felt justified in doing a spinal puncture, and the spinal fluid Wassermann test was found to be positive.

Dr. Pierce believed there is a chance for improvement in the classification of deafmutism, and suggested that syphilis as a cause should be in a class by itself. Where there is a history of deafmutism in the family there are deformities in the ear, of the cochlea, or of the nerve, or of the functional static apparatus. Consanguinity probably acts in the same way, producing deformities. In syphilis there are no deformities, but the operation of the law which demonstrates that there are three distinct elements of resistance to the nerves which pass through the auditory canal; the cochlear has the least, the vestibular has more, and the facial has still greater resistance. In cerebellar tumors the first part that goes out of commission is the cochlea, next the static apparatus and next the facial with facial paralysis, and the same is true of meningitis. In syphilis the function of nerves disappears in this sequence. The affection usually comes on from four to ten years after birth. In the cases that come on early Dr. Pierce's experience has been that the prognosis is good. He recalled four patients who had recovered their hearing under antisyphilitic therapy. If the cause of the condition is not ascertained in time the prognosis is worse, but in some cases strenuous syphilitic measures result in the establishment of considerable hearing.

This patient's mother had a four plus positive Wassermann reaction, and an elder sister gave a negative reaction. The father had not yet been tested. They expected to present the patient again after antisyphilitic measures had been carried out.

DR. WALTER THEOBALD presented a patient who formerly had a squamous cell carcinoma of the nose, which involved both nasal chambers and the sinuses. The same patient was presented three years ago and had remained well until recently following radium therapy. There was no nasal stoppage, and the tissue in the nose was fibrous and dense.

He had received four treatments with 25 mgs. radium in each nostril, at intervals of two weeks.

He was presented at this time because he had developed difficulty in swallowing; he could take only half a cup of liquid at a time and could swallow no solid food. Hoarseness had developed, with complete paralysis of the left vocal cord, as well as the arytenoid. There was marked edema and swelling in the region of the left tonsil, and the left half of the palate was swollen, soft and pushed downward. On four or five occasions the palate had been incised and one to two teaspoonfuls of pale yellow fluid withdrawn. The patient complained of some earache and headache, and there was tenderness over the left antrum. A skiagram of the chest led to the suspicion of metastatic growths in the mediastinum and recurrence of the malignant growth in the maxillary sinuses. The esophagus showed no involvement. Further treatment with radium was to follow.

**A Case for Diagnosis (Squamous Cell Carcinoma, Syphilis (?) ).**

DR. FRANCIS LEDERER presented a man, aged 58, with soreness of the left side of the mouth and some cough. A recent Wassermann test was reported negative. The soreness came on following the extraction of the last two left molars. There was no difficulty in swallowing, but the patient complained of soreness in the throat. Examination revealed an ulcer with slightly infiltrated edges, which spread upward over the palate and involved the cheek. It seemed to be a serpiginous process, with raised, papillomatous excrescences. Another Wassermann test was made and tissue removed for biopsy, but no definite etiology was established. A second biopsy suggested a lipoid affair. There was considerable pigmentation of the pharynx. Examination of the chest showed evidences of involvement and an adhesive pleurisy involving the diaphragm.

The patient was treated by surgical diathermy and went on to apparent recovery. Several months later he presented himself with a retro-pharyngeal process, with glandular involvement. The glands broke down and suppurated and the pus showed a hemolytic streptococcus. He received many applications of quartz light to the neck and pharynx. After the ulcer healed the patient was presented at the clinic on several occasions. The healed ulcer had the punched out appearance of a syphilitic lesion, but examination showed it to be a squamous cell carcinoma of the hypopharynx. The process in the palate may have been of the same type, but there was a possibility that there was a syphilitic involvement as well as carcinoma. The patient had been under observation since August, 1925, and at present the glands of the neck showed metastases.

**Atresia of Pharynx.**

DR. GEORGE T. JORDAN presented a complete atresia of the pharynx as the result of a thorough adenoidectomy and tonsillectomy. Following this the parents noticed that the boy did not breathe at all through the

nose. They returned to their physician and some adhesions were cut. When first seen by Dr. Jordan, two years ago, there was complete atresia.

Examination with the Holmes pharyngoscope showed the condition above the adhesions, and examination of the mouth demonstrated the complete atresia. The child was taken to the hospital, given a general anesthetic and the soft palate was freed from the postpharyngeal wall. A large heavy piece of dental rubber, stiff enough to keep the tissues well separated, and yet not stiff enough to cause sloughing at the angles of the wound, was placed between the soft palate and the pharyngeal wall, supported by two gauze strips brought out through the nose and tied, the nose being properly protected. The patient was able to swallow and to talk without any trouble. He was kept in the hospital for a week and then permitted to go home, still wearing the rubber. This was worn for three weeks more, after which it was removed.

It was exactly twenty-three months to the day at the time of presentation since this operation was performed. There had never been any closure of the wound, the child breathed freely through his nose and articulated distinctly. As almost two years had elapsed, Dr. Jordan felt justified in reporting this case and bringing it before the Society for observation.

#### **Mastoiditis With Meningeal Symptoms.**

DR. SAMUEL J. PEARLMAN presented a patient, aged 53, who entered the hospital October 21, complaining of pain in the left ear of six weeks' duration, spontaneous discharge of one week's duration, and weakness of the left side of the face that had persisted for three or four days.

Examination at that time showed the pulse and temperature to be normal; the tonsillar pillars and pharynx injected; a foul smelling, purulent discharge from the left ear, and a perforation in the inferior posterior quadrant. There was peripheral paresis of the left facial nerve and slight tenderness of the left mastoid tip. Blood count showed 7,200 leucocytes; the Wassermann reaction was negative; skiagram showed the left mastoid to be sclerotic. A spinal puncture gave a cloudy spinal fluid, under increased pressure, with 5,000 cells, polymorphonuclear leucocytes predominating. Smear and culture were negative.

A radical left mastoidectomy was performed on October 25 and a sclerotic mastoid found. The sinus and dura were exposed and found normal. Following the operation daily spinal punctures were performed and the cell count went down to 500. He also received one injection of polyvalent vaccine.

The patient complained of occasional headaches over the left temporal region, and on one occasion of "sticking" pain over the distribution of the left fifth nerve.

The fundi were negative; visual fields normal, and there were no neurologic findings aside from the left facial paresis.

**Mastoiditis With Meningeal Symptoms.**

DR. PEARLMAN also presented a boy, aged 13, who consulted him complaining of stiffness of the neck, soreness of the back, legs and sides and headache of four days' duration. He gave a history of discharge from the ear one year ago, which lasted for a few days, and recurred ten days before he consulted Dr. Pearlman. A tonsillectomy was advised and performed at that time. The earache was less severe following this operation, but a week later he noticed discharge from the left ear.

Examination showed the temperature to be 102° F., pulse 96, respiration 22. There was a bloody discharge from the left ear; the left mastoid was tender to pressure. There was a positive Kernig and Brudzinsky, a positive bilateral Babinski, a positive Chaddock, and rigidity of the neck. The labyrinthine examination was negative. The spinal fluid was under increased pressure, slightly cloudy, 3,200 cells per cm., showed extracellular diplococci and a pellicle on standing. On culture it showed the streptococcus hemolyticus. The fundi were negative.

A simple mastoidectomy was performed on September 9, 1927. The sigmoid sinus was exposed and a thrombosed vein was found; no pus. Subsequent spinal fluid findings had been within normal limits.

**Mastoiditis.**

DR. SAMUEL SALINGER presented a laborer, aged 20, who was first seen on November 17, 1927, complaining of pain in the head and chest, weakness, chill, fever, malaise, of six days' duration, and pain in the lumbar region, which had persisted for four days.

Examination revealed photophobia, a slight rise in temperature (99.8° F.), blood pressure 120/60, and a purulent discharge from the right ear, which the patient said had occurred at intervals for five years. The urine was negative, as was the blood and spinal fluid Wassermann reaction. The fluid was under normal tension, clear, the Pandy was four plus, and there were 560 cells per cubic millimeter, polymorphonuclears predominating. Gram positive, intracellular diplococci (pneumococci) were present in abundance. The patellar and Achilles reflexes were exaggerated, the Babinski was negative on both sides and the Kernig was greatly exaggerated.

Examination of the ear showed a purulent discharge, loss of the greater part of the drum, small cholesteatomatous masses and some granulations in the inner tympanic wall.

Diagnosis: Acute exacerbation of a chronic suppurative otitis media, with meningeal complications and cholesteatoma.

On November 18th, the right mastoid was exenterated, and proved to be sclerotic and apneumatic. The antrum was found in the usual location, of average size, filled with granulation tissue and cholesteatoma. A radical mastoidectomy was performed. Subsequent spinal puncture, three days after operation, showed 20 cells per cm., and negative cultures; the fluid was clear, under normal tension.

The interesting point in this case was that no communication was found between the mastoid antrum and middle ear with the cranial cavity. The meningeal infection was evidently by extension through communicating thrombosed veins. The patient made a perfect recovery.

DR. ALFRED LEWY presented the patient, whose case was described as septic meningitis at the November meeting. He had apparently recovered but still showed a spontaneous nystagmus on both sides and upward, and the Kernig was still positive on the left.

#### **Sarcoma of the Nose.**

Dr. Lewy also presented a girl, aged 11, who had a growth which filled the left nostril. Three radium emanation tubes of three millicuries each had been inserted. A few weeks later the growth had shrunk until it could be seen to be attached to the septum, when it was snared off. There had been no evidence of recurrence or metastasis. Microscopic examination showed the growth to be round cell sarcoma.

#### **SCIENTIFIC PROGRAM.**

##### **The Significance of Wave Length in the Therapeutic Use of Light (Illustrated).**

(ABSTRACT.)

W. J. BOVIE, professor of biophysics, Northwestern University Medical School, said he realized that he must talk to practicing physicians if he is to establish a liaison between his activities in the laboratory and classroom, and therapeutic practice. Without this liaison all of his research would drift out of the channels which lead to practical results. He could not speak in the language of clinical empiricism, for he had neither experience nor reading knowledge upon which to draw to enable him to tell how to treat this or that pathologic condition. The scientist must accept the empirical data as facts because they are empirical. Listening to the results he can only restate them in his own language of "as if," and reply, "That is interesting; it is as if this or that had happened."

At the meeting of the research committee of the American Sanatorium Association the ever recurring question was raised as to which is the best source of radiation for treating tuberculosis—the sun, the carbon arc, or the mercury vapor arc. He suggested that the simplest way to find out would be to distribute the cases in one of the larger sanatoriums and treat some with one source and some with another, so that by the end of a year one ought to be able to answer this question definitely. He was told that this was utterly impractical, for if twelve clinicians were to write up the progress of treatment in a single case there would be twelve different answers, and if they were working with several hundred cases the number of answers would be so great that it would be impossible.



to deduce any significant conclusions. It was also added that when all of the cases treated have been considered there is no substantial evidence at present that light has any curative value in tuberculosis. Another thing he learned was that if an attempt was made to divide the cases as he suggested, the psychologic effect on the patients would be so great as to outweigh any therapeutic effects which the exposure to light might have, for patients invariably have a belief in the light which has been most used at the particular sanatorium, and therefore have no faith in the curative powers of any other, even if used by the same clinician. The director of one tuberculosis sanatorium stated that one-third of the patients who could be successfully treated by ultraviolet light had laryngeal tuberculosis, and emphasized the necessity of combining local and general irradiation.

While he was perfectly willing to accept such empirical facts as seem reasonable, and which have been observed by men in whose judgment he has confidence, he thought he could easily quote from papers he had read, statements that convinced him, and he believed would convince his audience, that their authors were appallingly ignorant of the subject they attempted to clarify.

Empirically, light is used in two ways: First, a general radiation of the patient's body; second, a local application of ultraviolet light through the use of quartz rod applicators. The first method of procedure is supported by biologic facts of very general and broad significance. The second method of application possesses certain limitations which, he thought, have not always been known to the clinician, and because of this he was in doubt as to whether some patients had actually received the exposures which it was thought had been given.

That light has a profound influence upon growth and differentiation had been forcibly impressed upon Professor Bovie by his own experiments as well as by those of others. He described an experiment at Orono, Maine, where 250 chickens were divided into lots and raised under controlled conditions, which allowed the following conclusions to be clearly established:

Chicks raised under glass were lacking in vigor, gained only slowly in weight, and nearly all of them succumbed to rickets or leg weakness.

Chicks raised under glass, but which received fifteen minutes of irradiation daily from a quartz mercury vapor arc, were vigorous, free from disease, gained regularly and rapidly in weight, and matured sooner than chicks running freely in the sunlight. These observations were not only symptomatic but were supported by the evidence of skiagrams.

That the influence of light upon plants promotes differentiation of the tissues as well as growth, is clearly shown by the lack of differentiation and consequent weakness and simplicity in structure of plants raised in darkness. These differences are due to the presence

or absence of light derived from a very limited region of the spectrum, that lying between the end of transmission through glass and the end of sunlight.

Plants bend toward the light. This bending follows a very definite law, and experiment seems to show that a very small amount of a photoproduct, water-soluble, and hence diffusible through the plant, furnishes the agency by which the tissues on the lighted side of the plant become earlier differentiated, so that growth is restrained.

While this substance is formed demonstrably in plants, the product may be transmitted to animals as they feed upon the plants. It may be in this way that the cod gains such a substance and stores it in the liver, whence, therefore, comes the potency of cod liver oil. It seems clear that at least one reason why cod liver oil and ultraviolet irradiations do good in rickets is that they influence metabolism of calcium and cause it to be laid down in bone.

It must not be forgotten that, just as a photographic plate may be overexposed, this photo-product may be destroyed by over-irradiation.

The theoretical advantages gained through the use of quartz rod applicators are chiefly due to the fact that they gather rays of great angular dispersion, and lead this light of successive internal reflections to the point of application undiminished in amount, except as part of it may be absorbed by the rod. A secondary advantage lies in the fact that the light may be led by curved rods to places otherwise unattainable.

The practical employment of these rods is, however, hedged about by difficulties in controlling and directing the emergent beam of ultraviolet so that the physician may have confidence that he is getting a usable beam. Most, if not all, of the applicators on the market are undependable, and utterly fail to deliver the radiations that they are supposed to supply.

#### DISCUSSION.

H. J. HOLMQUIST, B. S., secretary of Council of Physical Therapy of the American Medical Association, said that a quarter of a century ago the advertising pages of medical journals were filled with advertisements of medicinal preparations, for which therapeutic claims were made that were even more startling than some of the therapeutic claims made today for electric heaters, similar to the ordinary bathroom heater. The most significant move in the warfare against this chaotic situation in the field of drug therapy was the establishment of a Council on Pharmacy and Chemistry by the American Medical Association, at the instigation of Dr. Simmons. Fraud and gross deception in drug therapy has been successfully combated until now reputable medical journals are free from flamboyant and preposterous advertisements of drug preparations.

Today there is probably as chaotic a situation in the field of physical therapy. During the World War it was shown that physical

therapy has a legitimate place in medicine, and since then the practice of this mode of treatment has extended tremendously. Not only are the merits of physical therapy being recognized by reputable physicians, but, unfortunately, cultists, barbers, masseurs, beauty parlor operators, Turkish bath proprietors, chiropractors and osteopaths have seized upon it as a profitable means of increasing their income. Physical energies have certain definite, known physiologic effects and perhaps other equally as definite, but as yet unknown, effects of a harmful nature. Therefore, irresponsible people, untrained in medical science and unlicensed to practice medicine, should be prohibited from diagnosing and treating disease by means of physical energies, for the same reason that such people are prohibited from practicing medicine and surgery.

Apparatus generating every conceivable form of physical energy is being advertised to the medical profession for therapeutic use. Probably much of the reputed therapeutic worth of these devices can be attributed to the nickel and enamel with which they are adorned. Physicians are being importuned by salesmen, absolutely untrained in medical science, to buy such apparatus. These men take it upon themselves to instruct physicians in the use of such apparatus and attempt to teach therapeutic applications. It is, therefore, not surprising that unwarranted therapeutic claims and absurd theories of the biologic and physiologic effects of physical energies are made.

Some of the manufacturers, realizing that physicians need training in physical therapy, established courses of one or two weeks' duration. Although some of these courses were good, they were woefully inadequate. A physician cannot acquire the requisite training in biophysics and in the application of physical energies to the treatment of disease in so short a period of time. A number of itinerant physicians are about in the land giving similar short courses, which are probably fostered by manufacturers for the purpose of promoting the sale of their apparatus. Heretofore, these courses could not be condemned too severely, commercialistic and inadequate as they were, for courses in physical therapy and biophysics were not offered by the medical schools. But an increasing number of medical schools are establishing courses in biophysics and physical therapy, and soon manufacturers will be unable to justify their courses on the ground that schools are not providing adequate training in physical therapy. The better manufacturers have already discontinued such educational work.

Innumerable books of more or less value have been written on physical therapy. Some of them have apparently been written with an eye to the royalty statement or the publicity returns rather than to the possibility of scientific criticism, by men whose qualification for the part of author appears to be that they have read a medical dictionary. Statements appearing in some of these books on the physics of apparatus for physical therapy are apparently culled

almost entirely from the advertising and descriptive matter published by a manufacturer. Some of the physical concepts appearing therein are wholly at variance with the concepts generally accepted by physicists, and are probably due to a desire to make physical science conform to absurd, preconceived theories as to the biologic effect of physical energies. Such books do not serve as trustworthy guides but only as wanton displays of the authors' pathetic ignorance or conscienceless disregard of the fundamental laws of physical science.

The present knowledge of physical therapy consists chiefly of unscientific statements of clinical experience and of therapeutic claims which apparently are not based on scientific observation. The extravagant therapeutic claims made for various physical energies by manufacturers are perhaps as much due to ignorance of the biologic effects of such energies as to a desire to sell apparatus. Later, when more scientific information as to the effects of such energies is available, it is possible that even more remarkable therapeutic claims may be warranted. However, before sound progress can be made in the science of physical therapy, this present mass of information must be critically examined and the true separated from the false. The present known merits and limitations of physical therapy must be clearly set forth in order that it may develop along sound lines.

To assist in placing physical therapy on a rational basis, the Council on Physical Therapy was created by the American Medical Association. The Council as now organized comprises physicists, physiologists, pathologists and clinicians. The general manager and secretary of the association, and the editor of the journal are ex-officio members of this council.

This council, having to deal with problems which lie in a field that has, as yet, been inadequately explored from a scientific viewpoint, has proceeded slowly and with conservatism. Without an exception, the manufacturers of apparatus have signified their desire to cooperate with the council in placing physical therapy on a scientific basis. Such cooperation by manufacturers and by the medical profession will greatly aid the council in solving the many problems with which it must deal.

Before physical therapy can develop along sound lines it must be divested of the unwarranted therapeutic claims and absurd theories with which it is now burdened. The council intends to do this by publishing a series of articles on the various branches of physical therapy, in which all that is definitely known of the value and limitations of these modes of treatment will be given. In this series will be articles on X-ray and radium, on heat, on light, on electrotherapy, on hydrotherapy and on massage, therapeutic exercise and mechanotherapy. Several of these articles are now completed and have been published in the journal. After publication in

the journal the articles will be collected and published in one or more books for the guidance of physicians.

Apparatus and methods for physical therapy will be considered by the council with a view to assuring the profession that a device advertised to deliver a particular energy will deliver such energy, and with a view to minimizing fire and accident hazards, and with a view to checking up the therapeutic claims made for such apparatus and methods. A set of official rules was adopted, to be followed in action on these devices. These rules were adopted primarily with a view to protecting the medical profession and the public against fraud, undesirable secrecy, and objectionable advertising in connection with the manufacture and sale of apparatus for physical therapy. These rules will also be of great service to manufacturers of such devices in standardizing their apparatus.

The reports of the council on devices and methods for physical therapy appear in that part of the journal devoted to the work of this council. Those devices and methods which have been found acceptable as conforming to the official rules will be described in a list of accepted physical therapy apparatus, which will be a book similar to "New and Nonofficial Remedies," published by the Council on Pharmacy and Chemistry. General reports on matters of importance to the medical profession and to the public are published from time to time by the council.

The council has not restricted itself to the consideration of therapeutic apparatus and methods, but will undertake the investigation of such matters as have importance to the medical profession and to the general public. For example, experiments were conducted by the council to determine the degree to which various materials advertised as pervious to ultraviolet rays will transmit such radiations and to determine the biologic effect of radiations filtered through such materials. The council report of this investigation has standardized this particular field of physical therapy. Not only has this report made available desired information, but it also has stimulated other scientific bodies to do work in this field. It is encouraging to believe that the statements and reports published by the council will always serve this twofold purpose, for by stimulating research and by interesting properly trained men in physical therapy the day when it will be on as rational a basis as pharmacotherapy is today will be materially hastened.

DR. J. GORDON WILSON, Chicago, said that among those practicing otolaryngology there is a lack of fundamental knowledge on the application of ultraviolet rays, and so they eagerly welcome information regarding the physical properties of these rays. No one is better qualified than Dr. Bovie to help to a clear understanding.

Dr. Bovie had stated that he had no clinical experience, and yet the facts he had brought out and the slides he had shown cannot but be of great value, and questions of interest to the clinician might well be asked of him.

The slides Dr. Bovie had shown illustrating the effects on plants of light rays raised in one's mind the question whether the action of electromagnetic rays on plants can be regarded as comparable to the effects on man, for there are present in plants elements not present in man which respond to light rays as well as to ultraviolet rays. The illustration he gave of the effect of a very short ultraviolet ray on ameba make one imagine that some time or other there will have to be differentiated at least two varieties of ultraviolet rays, as we are now differentiating varieties of X-rays.

Dr. Wilson thought there could be no doubt that a great amount of good had been secured in suitable cases from general irradiation of the body, but he had not found that local treatment had been very successful. He agreed with Dr. Bovie that before such local treatment can succeed we probably must have better quartz applicators.

Another point which puzzled him was the difficulty of determining when a patient becomes immune to ultraviolet rays and for how long a period should treatment be intermitted.

A good deal of attention has been given to the effect of those rays on otosclerosis. Some of these patients, after treatment, claim to hear better and, in fact, do hear the voice and whisper better, at least for a time, but in the majority the audiometer and tuning fork tests indicate that there is not much change. He was inclined to believe that here we are dealing with a general improvement in health and with it an improvement in hearing. In winter, with the foggy atmospheres of our large cities, ultraviolet rays are destined to be of very great service. In summer they can never supersede fresh air and sunshine in the country, especially if accompanied by exercise.

*Regular Meeting, Held on January 9, 1928.*

THE PRESIDENT, DR. GEORGE F. FISKE, PRESIDING.

**Drainage of Cisterna Pontis Lateralis in Meningitis (Case 2).**

(AUTHOR'S ABSTRACT.)

DR. ALFRED LEWY: A month ago I presented a case of purulent meningitis with recovery. In this case the outcome was not so fortunate. The patient was a female, aged 29. Both ears had discharged since childhood. In October, 1927, she complained of vertigo, ataxia, vomiting. A mastoid operation was performed, followed by relief of symptoms. Six weeks ago she had left facial paralysis. Since then she spent ten days in another city, where she was delirious, and under morphin. When I saw her there was wild delirium; stiff neck, positive Kernig; fever; purulent discharge from the left ear, mastoid scar with two fistulæ, left facial paralysis; totally deaf in the left ear; could hear conversation at three feet in right ear. The spinal fluid was turbid and under pressure.

A radical mastoid operation was done, uncovering the dura, middle and posterior fossæ, lateral sinus and Trautman's triangle, dissecting up dura from the posterior surface of petrosa to cisterna pontis lateralis, which was opened; on the way a small accumulation of pus was encountered and a xeroform gauze drain was inserted. The patient was more quiet, not requiring morphin, but she died on the fourth day after operation.

**Specific Labyrinthitis.**

DR. AUSTIN A. HAYDEN presented the case report of an Italian woman, aged 34, who was first seen on December 1, 1927, when she complained of severe headache, vomiting and discharge from the left ear. The vomiting and headache had been pronounced for four days. She was admitted to St. Joseph's Hospital and an extensive mastoid operation was performed. There has a history of recurrent suppuration in the left ear for the past nine years. She had two living children and had had three miscarriages. Her husband admitted having syphilis and taking treatment for it for eleven years. He stated that repeated blood and spinal fluid Wassermann tests on the patient were negative. The husband's acuity of vision consisted of an ability to count fingers at about six feet, his pupils were stationary and the optic discs almost snow white.

The patient's vision apparently was normal; the discs were normal, the pupillary reactions prompt, and the intraocular findings were negative. The mastoid operation revealed an acute exacerbation of an old, suppurative process that had dissected out most of the mastoid cavity and cells. The destruction extended almost to

the facial nerve, and under pressure in the atticus caused facial twitching. Examination of the spinal fluid showed a 4 plus Wassermann reaction, 44 cells and a sterile fluid. Antisymphilitic therapy was immediately instituted.

When the patient was first seen there was a marked rotary nystagmus to the right in the horizontal position, and a marked horizontal nystagmus with the head in the vertical position. About twelve days after the operation facial paralysis was noticed, which apparently became more marked. It was thought that this might be due to pressure from the dressings, or from the filling in of the granulation tissue.

She was discharged from the hospital on January 8, 1928, with the wound in good condition.

#### DISCUSSION.

DR. JOSEPH C. BECK said, in referring to the facial paralysis, it must not be forgotten, in cases of this type, that a swelling of the nerve from antisymphilitic treatment might produce the paralysis, but by following up the treatment it would probably clear up.

He asked if hearing and caloric tests had been carried out.

DR. HAYDEN stated that the hearing in the affected ear was for a whisper at eight feet, which he considered very good under the conditions that were present. No caloric tests were made because the patient was extremely nervous. The falling reaction was entirely to the left side when he saw her in her home the first time.

DR. GEORGE W. BOOT expressed the opinion that Dr. Hayden had failed to prove that this was a case of syphilitic labyrinthitis, as a syphilitic patient could have any type of labyrinthitis the same as any other patient.

DR. HAYDEN agreed that it might have been better to describe the case as mastoiditis occurring in a syphilitic patient, with semicircular canal disturbance. Under the antisymphilitic therapy she was making marked improvement.

#### Abnormality of Voice.

DR. DANIEL HAYDEN presented a patient whose voice had been normal up to the age of 14, who had been sent to him for observation because of marked bowing of one vocal cord and slight bowing of the other. The boy was to be turned over to Dr. Kenyon to determine whether or not it was a functional condition.

#### DISCUSSION.

DR. ELMER L. KENYON said that he had not obtained a sufficiently clear view of the larynx. What he saw that was unusual was a groove parallel with the border of the right cord, and a marked bowing of the cords in the anterior part of the larynx. The question for decision was whether the condition seen in the vocal cords was



primary, or, on the other hand, was secondary to a primary functional disturbance. The voice was high-pitched and strained, and the thyroid cartilage moved upwards with tension precisely as it does in the falsetto voice of puberty. Such a malaction of the extrinsic muscular system results in a bowing of the cords similar to that exhibited by the patient.

It was interesting to note that the trouble came on at the time of puberty and when the boy was living a rather unusual life in a soldiers' camp. Bearing in mind that at such times trouble with the talking function frequently occurs, and from similar conditions of nervous stress, there was a good basis for reasoning that the trouble might be functional. He felt that probably the trouble was primarily functional, but would study the case further.

#### **Diphtheria of the Middle Ear.**

DR. GEORGE J. MUSGRAVE presented a man, aged 24, who consulted him during the past summer because of a chronic, foul smelling discharge from the ear. The hearing was practically normal. The ear drum presented some thickening and complete detachment of Shrapnell's membrane. With a cotton applicator the debris was cleaned out, and this had to be repeated every six weeks. Skiagram showed slight change in the mastoid process. Microscopically the discharge resembled that given by accumulated leukocytes, but was yellow. Culture showed the Klebs-Loeffler bacillus.

#### **Dentigerous Cyst.**

Dr. Musgrave also presented a patient with a dentigerous cyst. He gave the usual history of a maxillary sinusitis, but on going through the sinus the cyst was found to be a separate compartment, not communicating with the nose.

#### **Management of Malignant Disease in Otolaryngology.**

DR. JOSEPH C. BECK read a paper entitled "Management of Malignant Disease in Otolaryngology."

#### **(AUTHOR'S ABSTRACT.)**

Dr. Beck reviewed his experiences with malignancies about the head and neck over a period of twenty-five years. He believed the discouraging outlook of the past has been replaced by a more hopeful attitude, due mainly to a newer approach in the attack on malignancies. Radical surgery, combined with "en block" dissections of the associated glands in conjunction with surgical diathermia, X-ray and radium, has changed the outlook and given more hope in combating this problem. Extensive radical surgery was stressed as far as possible by means of surgical diathermia. The objection to the use of surgical diathermia, namely, postoperative hemorrhage, can be obviated by prophylactic ligation of the external carotid or, if found necessary, a slow compression ligation of the common carotid. In the "en block" dissection of the glands the jugular was frequently sacrificed and occasionally even the common carotid.

The efficacy of laryngectomy for laryngeal carcinoma was stressed. The technic of surgical diathermia and the use of synergistic analgesia were described, as were some of the newer prostheses for remedying the postoperative deformity. The "open" treatment of the wound with radiotherapy and the careful check on the granulations by biopsy were also discussed. Emphasis was placed upon prevention of pain in hopeless cases by trigeminal nerve injections or gasserian ganglion resection.

## DISCUSSION.

DR. G. W. BOOT congratulated Dr. Beck on his results and agreed that diathermy in cases in which it is suitable is perhaps the best means for treating carcinoma of the throat, nose or head, but even this is not uniformly successful.

Regarding the five-year period of nonrecurrence as indicating a cure of cancer, he expressed doubt as to whether cancer could ever be considered as certainly cured. It is not the simple thing that some seem to think, or the cause of cancer would have been discovered long ago. He does not think that cancer is hereditary, or it would appear at a definite time of life just as other things do that are hereditary; instead it has appeared at all ages, from infancy to extreme old age. He does not believe that cancer is born in the cell or in the ovum or in the chromosomes. It is not a cell rest, nor is it the result of irritation purely. If it were, most of the men in Chicago should be having cancer of the face, for who does not continually irritate his face while shaving? He has on record two cases where cancer followed on one single act of trauma. One man accidentally cut his lip with a cleaver and cancer followed. Another was struck on the cheek by a flying chip of wood and cancer developed on the little wound. Cancer can be produced in rabbits and in mice by painting their skin with coal tar. Some slow chemical or biochemical change takes place in their bodies, and particularly in the structures which develop from epiblast, that prepares the way for cancer; then comes the irritant, which is often a chronic irritation but may be a single act of irritation, and a cancer develops. Thus it is that the smoker's pipe causes cancer to develop on his lip. If a clean and complete operation is done for cancer of the breast the patient may see no sign of return for five years, but later the disease can return, because the system has already been prepared and only awaits a suitable irritation for the cancer to reappear. This is probably why so many cancers of the breast recur, either locally or at some distant point. It is not reasonable to suppose that a metastasis occurred at the time of operation or before and lay dormant for five years. Local recurrence may be due simply to the irritation of scar tissue in a previously sensitized patient.

Dr. Boot has almost never seen a cancer of the mouth or throat in a patient who kept his mouth clean. As a rule, such patients have perfectly filthy mouths. They appear to have never used a tooth-

brush. It may be possible that the toxins from these filthy mouths may be the sensitizing agent. Whatever it is, it is a slow process, which in the average patient extends over many years. In the mouse the tar must act for about two months before cancer occurs. In the rabbit it takes several months.

Wallin of the University of Colorado and Buchner of Greifswald have recently done considerable work on symbionticism. They find that in many forms of animal life bacteria live within the cells of the animal during its whole life without doing harm. In fact, that the animal really seems to be a combination of animal cells and bacteria. It seemed to the speaker that further knowledge along this line may explain the development of cancer.

DR. MICHAEL GOLDENBURG thought Dr. Boot's statements worthy of consideration, but asked how he accounted on that hypothesis for malignancy under one year of age, particularly the intraocular malignancies. McCarrison, on his return from Thibet, where he spent two years, stated that he did not see a single case of malignancy, but as he again approached civilization the malignancies appeared.

Dr. Goldenburg was inclined to think that Dr. Boot was correct in his assumption that no malignancy is ever cured. If a malignancy could be recognized when the first cell divides one might be able to accomplish something, but after division takes place the new cells do not always remain at the point of origin but may locate anywhere. It is probably true that the nature and character of the new cell, the environment and tissues in which it was born play an important part in its growth and ability to spread to other parts. McCarrison's observations are interesting in that they tend to add weight to the theory based on a defective metabolic chemistry.

Dr. Boot, replying to Dr. Goldenburg, said the explanation for the very early cases of malignancy was probably based on the fact that some individuals are more likely to develop cancer than others. Cancer is not born in one, but some individuals have a predisposition to the disease. The malignancies under one year of age are principally sarcomas, which develop from a different type of cell, although cases of carcinoma and sarcoma both are on record, and one type of growth changes into the other in some instances.

DR. BECK (closing) thanked Dr. Boot for his discussion, and said that the Society for the Control of Cancer believes five years of life following operation constitutes cure. A patient may live for five years and die of an intercurrent disease. He called attention to the fact that cancer has never been known to develop in a blood vessel or in cartilage, probably because there is no epithelial structure there. A Polish observer, Adamkovitch, has made a serum from cartilage and arterial walls and uses this for injection purposes, just as Fichara some years ago took liver and embryonal tissue mixed with cancer tissue and injected it into cancer patients, with marked results in stopping the growth.

Dr. Beck felt that irritation has a great deal to do with the development of cancer. Animal experiments have little bearing on cancer in man, but irritation in animals can produce epithelial growths. Chimney sweeps develop cancer very frequently, and many other things prove the irritation theory, either from bacteria or something else. Dr. Emil Beck has recently shown cases of ten to fourteen years' duration, and he felt sure that relief can be obtained for comparatively long periods, if not cure.

Dr. Boot repeated his assertion that irritation alone will not produce cancer, but that it acts in combination with some other element.

DR. GEORGE F. FISKE asked Dr. Boot why he had been able to produce cancer in white mice and not in white rats. If there was this exemption in animals, why could it not occur in human beings as well?

Dr. Boot replied that he thought he did not keep up the work long enough with the white rats to obtain this result, but that he could have produced cancer had he continued the experiments longer.

*Regular Monthly Meeting, Held on February 6, 1928.*

THE VICE-PRESIDENT, DR. FRANK NOVAK, PRESIDING.

SCIENTIFIC PROGRAM.

**Alteration in Eighth Nerve Activity Produced by Rapid Blood Pressure Changes in the Normal Human.**

J. FRANK PEARCY, Ph. D., and DR. DANIEL B. HAYDEN presented a paper entitled "Alteration in Eighth Nerve Activity Produced by Rapid Blood Pressure Changes in the Normal Human."

In clinical hypertension there is frequently a decrease in auditory acuity for the higher (but not the highest) tones. Such patients commonly complain of tinnitus aurium and vertigo. That these symptoms are caused by the high blood pressure per se is a gratuitous assumption. It is equally likely that the pathologic structural changes associated with the hypertension produce the symptoms. In some instances hypotension has been associated with vertigo, tinnitus and a similar hearing defect. These symptoms are found occasionally in severe anemias.

One is tempted to speculate upon a common cause for the symptoms in the three cases. Such a cause might be an insufficient oxygen supply to the internal ear. There is a decreased oxygen supply in severe anemias. In marked hypotensions there is also a considerable decrease in oxygen supply. Hypertension is frequently associated with arteriosclerosis. Arteriosclerosis of the arteries leading to the internal ear could diminish the blood supply and thus the oxygen supply to the internal ear, as it does to the legs in intermittent claudication and to the heart in angina pectoris. But, presumably, a diminished oxygen supply should affect the entire auditory scale and not the higher tones alone.

METHODS.

A large model Western Electric (I-A) Audiometer and the usual rotation methods were used. Increased blood pressure was obtained at first by the use of ephedrine. It was not used after a few experiments because of the unpredictability of its effects. In some cases  $\frac{3}{4}$  gr. raised the pressure 40 mm. Hg. In other cases  $1\frac{1}{2}$  gr. raised it but 10 mm. Hg. Epinephrin injected subcutaneously and massaged, as recommended by Luckhardt, proved entirely satisfactory. The blood pressure could invariably be raised 50 mm. to 80 mm. Hg. in a very short period. It remained at a constant high level long enough for satisfactory labyrinthine tests to be made.

The blood pressure was lowered by use of sodium nitrite in from 3 gr. to 5 gr. doses.

## RESULTS.

**Auditory.**—A decrease in blood pressure from a normal of 128 mm. to 100 mm. Hg. caused a considerable impairment of hearing, whereas, an increase from 128 mm. to 160 mm. Hg. caused a considerable increase in auditory acuity. In the instances of both decreased and increased blood pressure the auditory changes were detected only in the upper tone limits. Perception of the very highest tones was not modified except in a few cases of lowered blood pressure. Results from typical experiments follow:

**Vestibular.**—Raising the blood pressure increased the vestibular responses, whereas lowering the blood pressure decreased the response. Typical responses were as follows:

1. Normal blood pressure 124 mm. Hg.

No spontaneous nystagmus or past pointing.

Rotation to right 10 times in 20 sec. After nystagmus, moderate amplitude, duration 20 seconds.

Rotation to left 10 times in 20 seconds. After nystagmus, moderate amplitude, duration 21 seconds.

Rotation to right 10 times in 10 seconds. Past pointing, right hand 8", no drifting.

Rotation to right 10 times in 10 seconds. Past pointing, left hand 6", no drifting.

Rotation to left 10 times in 10 seconds. Past pointing, right hand 8", no drifting.

Rotation to left 10 times in 10 seconds. Past pointing, left hand 6", no drifting.

Fifteen minutes after subcutaneous injection of  $\frac{1}{2}$  cc. 1/1000 epinephrin.

Blood pressure 160 mm. Hg.

Rotation to right 10 times in 20 sec. After nystagmus violent, duration 28 sec.

Rotation to left 10 times in 20 sec. After nystagmus violent, duration 28 sec.

Rotation to right 10 times in 10 sec. Past pointing, right hand 13", drifting to right and to right angle and upward.

Rotation to right 10 times in 10 sec. Past pointing, left hand 12", drifting to right to right angle and upward.

Rotation to left 10 times in 10 sec. Past pointing, right hand 12", drifting to left to right angle and upward.

Rotation to left 10 times in 10 sec. Past pointing, left hand 13", drifting to left to right angle and upward.

2. Normal blood pressure, 122 mm. Hg.

No spontaneous nystagmus or past pointing.

Rotation to right 10 times in 20 sec. After nystagmus moderate duration 21 sec.

Rotation to left 10 times in 20 sec. After nystagmus moderate, duration 20 sec.

Rotation to right 10 times in 10 sec. Past pointing, right hand 8", no drifting.

Rotation to right 10 times in 10 sec. Past pointing, left hand 6", no drifting.

Rotation to left 10 times in 10 sec. Past pointing, right hand 8", no drifting.

Rotation to left 10 times in 10 sec. Past pointing, left hand 6", no drifting.

One and one-half hours after taking 3 gr. sodium nitrite per os.

Blood pressure 100 mm. Hg.

Rotation to right 10 times in 20 sec. After nystagmus, very small, duration 8 sec.

Rotation to left 10 times in 20 sec. After nystagmus very small, duration 7 sec.

Rotation to right 10 times in 10 sec. Past pointing, right hand 1", to right.

Rotation to right 10 times in 10 sec. Past pointing, left hand 1", to right.

Rotation to left 10 times in 10 sec. Past pointing, right hand, touches.

Rotation to left 10 times in 10 sec. Past pointing, left hand, touches.

#### DISCUSSION.

It is conclusively demonstrated that a lowered systolic blood pressure produces symptoms similar to those found in clinical hypotension but that a raised systolic blood pressure produces symptoms opposite to those observed in clinical hypertension. To explain these hypertensial labyrinthine symptoms one must look to something other than the raised systolic pressure.

We can conceive of no way in which systolic blood pressure changes could cause this alteration of acuity of hearing except by an alteration in the vascular supply of that portion of the cochlea where upper tones of the scale are heard. By an analogy from a study of the effects of systolic blood pressure changes on the vessels of the optic fundus one would expect that when vasoconstrictors are administered the arteries of the ear are constricted, and when dilators are used dilatation of these arteries results. We have no explanation to account for the alteration in vestibular activity produced by systolic blood changes.

#### CONCLUSIONS.

1. A sudden lowering of a normal systolic blood pressure by 20 to 30 mm. Hg. produces an auditory impairment of 15 to 20 per cent. A sudden raising of normal systolic blood pressure by 20 to 40 mm. Hg. produces an increased auditory acuity of 10 to 15 per cent in the higher tone scale between 512 and 8192 double vibrations, the lower and the higher tones being unaffected.

2. A similar lowering of the blood pressure decreases the duration of after nystagmus 60 to 70 per cent, and past pointing 75 to 85 per cent. A similar raising of the blood pressure increases the duration of the nystagmus 10 to 20 per cent and past pointing 30 to 50 per cent.

## DISCUSSION.

DR. AUSTIN A. HAYDEN considered this subject of great importance, and thought if the experiments did nothing else than point the way to the relief of seasickness they would be well worth while. He was much surprised to find, on his recent trip to Europe, that the chief surgeon on the S. S. Roosevelt had found hypodermic injections of sodium nitrite the most useful and effective drug he had used for this purpose, and thought this an interesting coincidence. He was glad to learn that one patient suffering with tinnitus had been treated by Dr. Percy and would be interested to learn what results could be expected from the use of the drug in this disorder.

Dr. Hayden asked how long the blood pressure would remain elevated after the use of adrenalin and ephedrin.

He had been astonished in talking with the ship's surgeon to find that so small an amount of investigation has been carried on in regard to seasickness, and thought a good field for research could be gained if the various steamship companies and the surgeons on ocean liners would be interested in the matter.

DR. ARTHUR M. CORWIN expressed his interest in the subject, and thought the next step in the experiments should be to take a series of patients who became seasick and investigate their blood pressure with a view to determining whether patients with high blood pressure are more likely to be seasick than those with normal pressure, thus deciding whether there is a relationship between blood pressure and seasickness.

DR. FRANK NOVAK asked if any nitroglycerin had been used in the experiments.

DR. DANIEL B. HAYDEN said the effects of epinephrin lasted several hours. Clinically the symptoms of hypertension and hypotension are exactly the same. When they started this work they did not expect to find any variations of hearing by increasing or diminishing the blood pressure.

They had not used nitroglycerin because their work had been done on humans. The maximum effect from the nitrite was obtained in about thirty minutes.

DR. HARRY POLLOCK said he had taken the blood pressure in many cases following the use of one-half cubic centimeter of epinephrin, and the rise lasted only three to five minutes. The pressure went up very rapidly but fell just as quickly, and according to the physiologists the same was true of adrenalin. He felt sure that no relation existed between blood pressure and seasickness.



DR. J. FRANK PEARCY said Dr. Luckhardt had found that by the subcutaneous use of epinephrin the blood pressure could be kept up for many hours—until the following day, while if given intravenously it caused a sudden rise and an equally sudden fall, as Dr. Pollock had stated.

**The Relationship of Orthodontia to Otolaryngology.**

DR. BURNE O. SIPPY, by invitation, presented a paper on "The Relationship of Orthodontia to Otolaryngology."

(AUTHOR'S ABSTRACT.)

There are definite functions governing the normal development of the human denture. A perversion of such functions causes malformation of the face and jaws.

The mandible performs two important functions, namely, mastication and respiration. At birth all of the teeth, except the second and third permanent molars, have begun to develop. In the upper jaw they occupy almost all of the space to the floor of the nose and orbit with little indication of maxillary sinus. Each tooth germ is enclosed in a crypt, a wall of cribriform plate having spicules of cancelous bone. As the tooth develops pressure is created and the crypt wall is pushed backward through cancelous bone. The growth of the tongue and the organs of the mouth exert pressure from the lingual. The muscular forces from outside namely, the muscles of mastication, deglutition, respiration and expression, act as mechanical stimuli to produce a reaction of connective tissue cells, resulting in growth. Growth of the crown causes absorption of the roof of the crypt as the tooth moves occlusally cutting through the overlying soft tissue. As the tooth moves occlusally the bone grows with it, forming the alveolus. The thickness and the length and the width of the jaws increases as the teeth grow outward and forward and occlusally. The maintaining of the normal relation of the jaws to each other, the development of the maxillary sinuses, the nasal passage and the septum depend upon the balance of muscular force. A lack of these normal forces or a change in the direction of these forces will entirely change the development of the bones of the face and jaws and result in malocclusion. This condition may be complicated by a mutilation, as in the involvement of congenitally absent teeth, extracted teeth, cleft palate and harelip, including the nose, fractures of the mandible or maxilla, postoperative conditions following the surgical removal of tumors, etc.

Orthodontia is that science which has for its object the correction of malocclusion. Occlusion, the basis of orthodontia, is the normal relation of the occlusal inclined planes of the teeth when the jaws are closed. Malocclusion is the perversion of the normal. The practice of orthodontia has to do with the correction of the malocclusion of the teeth and malformation of the related bones of the face and jaws. The term mesial means toward the median

line; distal is the opposite term and might imply to you, posterior.

The first permanent molar is considered the key to occlusion. It performs many important functions in the development of the denture, the more important of which are that it determines the length of the bite of the adult denture; it governs the position of teeth mesial and distal to it; it is the most constant of the teeth in taking its normal position and it is most constant in its time of eruption and, therefore, has been termed the six year molar. For the reasons mentioned and others, it is the basis of the classification of malocclusion.

According to Angle, all malocclusion is divided into three groups: Class 1 malocclusion is characterized by normal mesiodistal relation of the jaws, as indicated by that normal mesiodistal position of the first permanent molar. Both jaws are equally underdeveloped, being short and narrow; or rarely, both jaws may be equally overdeveloped, being wider and longer than normal. About 70 per cent of all malocclusion falls in Class 1. Class 2 is characterized by a distal relation of the lower arch as compared with the upper arch, either unilateral or bilateral. It is characterized by a long, narrow upper arch with an underdeveloped mandible. It may or may not be associated with mouth breathing. About 25 per cent of malocclusion falls in this class. Class 3 is characterized by a mesial relation of the lower arch, either unilateral or bilateral. The mandible is overdeveloped, usually longer and wider than normal, and the maxilla is underdeveloped, being short and narrow. In medical terminology, this condition is called a prognathous jaw, and about 5 per cent of malocclusion falls in this class. Dental arches that are short and narrow will have a high and narrow palate. The floor of the nose will, therefore, be narrow, the sinuses underdeveloped and the teeth will be crowded and protruding. The tongue will not take its normal position in the roof of the mouth and will not exert its normal pressure upon the lingual surface of the teeth and the bone, but is carried in the floor of the mouth inert. The mandible sags, the lips and cheek muscles are functionless and there is a lack of muscle tone from within and outside the denture.

With this brief sketch of malocclusion and the characteristics of the bone formation, you may readily note that a large percentage of malformation involves the underdevelopment of the maxilla in width and length. This fact I believe to be of interest to the otolaryngologist. We are both treating children and are dealing with the problem of establishing a normal function insofar as it may be possible. When a child is presented to the orthodontist, we include in our case history such findings as follows: Is there any history of nose and throat trouble? Has there been any operations for adenoids and tonsils or other nose and throat operations? If so, by whom, when and where performed? What is the nose and throat condition now? Is normal respiration possible? If this is not found to be so, the patient is referred to the otolaryngologist for examina-

tion and for such treatment and operation as may be necessary to clear the nose and throat conditions. The orthodontist keeps a record of the height and weight of the patient at intervals during the entire period of management and observation. It is frequently necessary to refer the patient also to the pediatrician, the dermatologist, and the internist to improve general physical conditions. We are much concerned in having normal metabolism if such is possible.

Nose and throat conditions, the habit of mouth breathing, the sucking of the thumb, lips, tongue and other like habits fall toward the end in the scale of etiology of malocclusion in point of frequency. Yet in any given case of malocclusion when such is the principal etiologic factor of the disturbance in balance of the normal forces it becomes the most important factor, not only in the management of the malocclusion at the time, but for the future development of the face and jaws and, in fact, the entire individual.

We are constantly asked the question, "When is the proper time to treat malocclusion?" This may be answered in general terms, that malocclusion should be treated as early as it is recognized. There are certain periods of growth in the jaws which should be considered. When possible the management should be completed by the time the growth of the jaws is completed, which is at the age of fourteen, except for the third molars. Orthodontia management may be completed in periods of a few months for each period of growth. I do not mean to infer that malocclusion cannot be managed after fourteen years of age or managed successfully, but the period of treatment is longer and the total time of wearing appliance may require two to three years. The ideal time is during the periods of growth when the osteoblastic and osteoclastic cells are most active.

The deciduous denture unless mutilated, as in cleft palate and harelip, is usually normal at birth and unerupted teeth are in normal position. However, at the age of two, three or four, there may occur an underdevelopment or overdevelopment of the mandible in respect to the maxilla which indicates a beginning Class 2 or Class 3 malocclusion. This may mean hypertrophied adenoids or tonsils or both. Either of these conditions should be treated first by the otolaryngologist. The removal of adenoids and tonsils may in some instances prevent the establishing of a malocclusion, but if the malocclusion is established, the clearing of the nose and throat will not in itself correct the underdevelopment or overdevelopment of the bones involved. Either of these conditions may be and should be treated by the orthodontist before the absorption of the deciduous roots has taken place in the physiologic transition from the temporary to the permanent denture.

The next period of development is at the time of the eruption of the first permanent molar and the eight incisor teeth, at about six years of age. If functions and development have not been nor-

mal, the first permanent molars will not occlude normally. The same forces governing the normal development of the bones are equally powerful in increasing the inharmony which takes an impetus at this time. This is a very important time for management by the orthodontist.

In each succeeding period of growth—i. e., the eruption of the bicusps, the cuspids and the second permanent molar, the inharmony, if not previously managed and normal influences established, continues with increasing symptoms in deformity and inharmony until the maximum has been reached. The maxillary and other sinuses involved and the nasal passage will have a minimum development rather than their normal development, thus making it physically impossible for the individual to perform the normal functions of normal respiration and mastication. (Lantern slides.)

#### DISCUSSION.

DR. GEORGE J. DENNIS expressed his appreciation of this able and interesting paper, and thought the effect of irregularities of the teeth on the nose and throat was definite. He did not see the relationship between respiration and the function of the jaw in normal occlusion, but in an abnormal development this was marked. The question of irregularity of the teeth should be considered before birth, for dentists often see a child with its father's large teeth and its mother's small mouth. Prenatal influences have much to do with this.

There are three definite classes of malocclusion and of occlusion: First, a short and fairly wide jaw, both above and below, with fairly good occlusion. Second, the projection of the incisor teeth, with narrowing of the upper jaw and widening of the mandible. Third, protrusion of the lower jaw with the incisors closing over the upper incisors. This, he thought, has a certain effect on the nose and throat. In the first class one often finds a hypertrophic condition of the nose and throat; in the second, a narrowing of the nares and compression of the maxillary sinus so that they extend below the floor of the nose; in the third, there is very apt to be atrophic conditions, with widening of the nose. Especially in Classes 2 and 3 there is an effect on the laryngeal and pharyngeal mucosa, probably most marked in Class 2, and in these cases there is usually shortening of the upper lip. When this condition is present there is likely to be a pharyngitis and an intractable laryngitis until the occlusion of the teeth is corrected.

First teeth are usually regular, but with the eruption of the sixth year molars there is departure from this regularity, which is caused in most instances by caries of the first and second deciduous molars.

DR. EDWIN MCGINNIS cited a case of marked malocclusion of the incisors, high arched palate and sunken nares as the result of destruction of the soft palate and shrinking of the scar tissue, resulting in mouth breathing. In an operation for adenoids the soft

palate had been removed. Dr. Brophy attempted to remedy the condition, without much change in the end result. This child, now aged twelve, had a chronic infection in all of the nasal accessory sinuses, and almost total closure of the nasopharynx.

DR. ELMER L. KENYON believed one important purpose of the paper to be an effort to get cooperation in the matter of etiology between the rhinologist and orthodontist. There is an immediate relationship in the development between the nose, jaws and teeth, and an intimate joint study should be carried out by the rhinologist, the orthodontist and other departments of medicine. Dr. Sippy had stated that the projecting upper jaw resulted when the normal upward pressure of the tongue against the palate was made impossible by the dropping of the lower jaw for mouth breathing. This occurred usually because of adenoids. If this were true, it was time for the two specialists to work together early enough by removal of adenoids, so that this deformity could not occur; but he could not understand how the light pressure of the tongue as normally exerted could have such a marked effect as Dr. Sippy thought on the formation of the jaw. Recently his attention was called to experiments on two young monkeys to see what would happen if in one animal vitamin "B" was removed from the diet and in the other it was retained. The monkey receiving the vitamin "B" developed well, had rounded features, and the other became extremely atrophic, was small and had irregular teeth. He wondered if the partial absence of vitamin "B" might not account for some of the irregularities seen in human dental occlusion.

Referring to the fact that in one instance of cleft palate with malocclusion, the patient improved in speech as the malocclusion was corrected, Dr. Kenyon expressed the opinion that all of the articulative defects present in malocclusion can occur where there is perfect occlusion and a perfect palate. Speech is fundamentally a matter of intelligence. He sees almost no deformities with malocclusion in which there is a speech disorder which cannot be corrected without correcting the irregularity of the teeth. The malocclusion serves not so much to prevent normal articulative development as it does to discourage the child from making sufficient effort to overcome the not impossible handicap of irregular teeth.

DR. AUSTIN A. HAYDEN expressed his great interest in Dr. Sippy's remarks, and said that many patients are operated upon for the relief of mouth breathing. Most of the operations are successful, but some fail to restore nasal breathing, not because the operation is not well done but because some of the defects Dr. Sippy had pointed out were present and their importance was not realized. To prevent the chagrin of an unsuccessful operation there was a definite field for cooperation between the otolaryngologists and orthodontists, as the essayist had contended. In addition to the pressure of the tongue being a factor in the spreading of the upper

jaw, he believed there was an additional factor in the forcible pressure between the maxilla and mandible in chewing.

The first lesson to be learned from the paper, and the first benefit to be obtained was the early recognition of these cases by the otolaryngologist, before they are operated upon. A statement by him to the patient or the family that the mouth breathing will probably not be corrected by removal of obstruction alone, but must be followed or perhaps preceded by correction of the deformities, should always be made.

Dr. Hayden was particularly interested in the effect that the widening of the upper jaw has upon the widening of the nose, particularly in the region of the posterior nares, and believed that many failures in operating for the correction of mouth breathing are due to the fact that the width of the posterior coanal openings is not measured before the operation is attempted. The size of this opening is fully as important as a deflected septum or an obstructing turbinate. If a small coanal opening was discovered and there was a chance for widening the upper jaw, thus correcting this defect, the orthodontist had brought something to the profession they had not had before.

DR. BURNE O. SIPPY (closing) said that orthodontists do not know all that they would like to know regarding the development of human anatomy, of the physiology of respiration, of diet or of endocrinology. Not enough is known about these things at present to permit of clear cut statements, and the medical profession must do more research work along these lines, as they are trying to encourage orthodontists to do along the lines of a better understanding of deformity. He thought there was quite good evidence to show that the theory of the large teeth of the father and the small mouth of the mother as a cause of malocclusion has been exploded.

The question of Dr. Kenyon in regard to the amount of force of the tongue against the palate was not answered. This pressure is present to a greater or less degree all the time when one is breathing through the nose. The more vigorous and continuous the breathing the greater is the pressure, but the moment the lips are opened the tongue drops to the floor of the mouth and is inert.

Referring to the remarks of Dr. McGinnis, he said they not infrequently see cases in which the uvula has been removed with the adenoids, and it is necessary to try to restore the speech and other functions as well.

*Regular Monthly Meeting, Held on March 5, 1928.*

THE PRESIDENT, DR. GEORGE F. FISKE, PRESIDING.

**Presentation of Cases.**

DR. JOSEPH C. BECK presented a man, aged 29, on whom a laryngectomy had been done, who had interesting histopathologic findings of typical adenocarcinoma. The microscopic diagnosis made by Dr. Hektoen revealed a squamous cell carcinoma. Several pieces of tissue had been removed by suspension laryngoscopy for study and all proved to be adenocarcinoma. The man showed a gain in weight of twenty pounds in five weeks since the operation. The wound healed very rapidly. The feeding tube was allowed to remain until the esophagus closed, in two or three weeks. Dr. Beck thought the most interesting things about these cases were that the patients remain alive and that speech is resumed. It gave the patient the greatest encouragement when he heard Dr. Salinger's patient speak. There has been great progress in handling these cases. They have an esophagobuccal voice which is never perfect, but certainly is a great help to them. This man was a foreman in a factory, and had to speak quite plainly, and the case was best adapted for an artificial larynx.

DR. SALINGER presented a man, 40 years of age. In February, 1927, he presented himself with a neoplasm of the larynx, with clinical indications of carcinoma, with infiltration and fixation of the right half of the larynx and beginning involvement of the right side. A biopsy was reported to be a squamous cell carcinoma. A two-stage operation was attempted; the first stage consisted in skeletonizing the larynx and shutting off the mediastinum. He developed a terrific infection of the neck that resulted in sloughing of the tracheal sutures and loss of tissue. This lasted five weeks and the carcinoma extended into the neck, involving some of the glands. They thought that they had nothing to lose, so removed the larynx, the overlying skin in the midline, a number of attached glands and two inches of the anterior wall of the esophagus. The case seemed practically hopeless. The carcinoma had eroded through the laryngeal box into the tissues of the neck, involving overlying muscles, thyroid, isthmus and cervical glands. Histologic examination of the specimen confirmed the original diagnosis. They fed the man according to the MacKenty technic, through a feeding tube passed through the nose. At the end of three weeks, the neck wounds being about closed, they removed the feeding tube and the patient was able to swallow. He had gained rapidly in weight, reaching his normal weight. He apparently had learned to speak instinctively, producing a low guttural voice, which could be heard

across the room, and had no difficulty in making himself understood. Dr. Salinger thought the voice would improve.

The important features of the case were, first, that it was extrinsic, and, second, that after a bad start they went ahead with the work and obtained a good result.

DR. AUSTIN A. HAYDEN: The first case was presented to emphasize again the fact that a blood Wassermann reaction is not always infallible and that sometimes a blood Wassermann reaction will be negative when the spinal fluid test is positive. This woman, aged 36, presented that fact. The X-ray suggested the presence of a pituitary growth or a growth in the pituitary region. She complained of tubular vision and intense headache and with the spinal fluid ++++ there was the possibility that the growth was a gumma and that operative interference would be necessary.

The next case was that of a boy, aged 11, with a deformed upper jaw, of which Dr. Burne O. Sippy had made some casts. This boy was brought in because of the fact that he had very poor nasal respiration and could not breathe with the mouth shut. The front of his nose was well formed, the septum straight and the turbinates not obstructive. The tonsils had been taken out very cleanly. He was brought in with the idea of making a cast of the posterior choanal openings. A large amount of adenoid growth was found there. The posterior nasal openings were found to be very large, showing that even a mouse-toothed individual with high arched palate may have sufficiently large openings from the nose into the throat behind. Wax casts of the patient's nasopharynx were presented. The size of the posterior nasal openings was measured by the width of the mould at the level of the lower margin of the septum. An empyema of each maxillary antrum, together with the large adenoid remnant, were the cause of the child's persistent mouth breathing. With the clearing up of these conditions, the boy breathed very well through his nose. The symptoms for which he came in were entirely relieved, but from Dr. Sippy's casts it was obvious that he needed some orthodontal work. The case was presented to show that close cooperation between the otolaryngologist and orthodontist is advisable.

#### DISCUSSION.

DR. BURNE O. SIPPY said he did not have an opportunity to make a close analysis of this case, but it showed a normal development and growth in the mandible and presented a Class 3 malocclusion, a mesial occlusion, with arrested development of the entire maxilla. There was impaction of teeth in the upper arch with lingual occlusion, the upper anterior teeth being inside the lower arch instead of overhanging the lower arch. In taking the two uppers and putting them with the incisal edges together, this boy, at 11 years of age, showed the exact arch formation of a normally devel-



oped boy of five. He was not prepared to say how much the condition Dr. Hayden mentioned had been the result of this condition. He thought it would be possible to get bone growth there by orthodontia stimulation which would bring it up to normal development, and was quite sure that this would have some influence on his other troubles.

DR. HARRY POLLOCK called attention to some articles of Shea of Memphis, which showed bony development following clearing up of the maxillary sinuses. Shea showed some cases in which clearing up of the sinuses developed the whole maxilla, proving that these cases, without further stimulation, will develop if one eradicates the infection. This boy was a little older than Shea's patients, who were six and seven years of age, but the articles showed them again at nine, ten and eleven. Dr. Pollock thought there was no question that the orthodontist can be of great help, but after clearing out the sinuses the normal development which had been arrested by infection would occur.

DR. AUSTIN A. HAYDEN (closing) thought this child was beyond the age where restoration of nasal breathing and clearing of the sinuses would straighten out the teeth. From the position in which the child's front teeth were directed, with the deciduous teeth, that might be true, but in this case he believed the help of the orthodontist was absolutely necessary.

SCIENTIFIC PROGRAM.

**Total Paralysis of the Vocal Cord.**

DR. J. HOLINGER presented a paper on "Total Paralysis of the Vocal Cord."

(AUTHOR'S ABSTRACT.)

Total paralysis of the vocal cords is always a serious proposition. Fortunately it does not happen very often, but a few years ago I saw three cases of it at the same time, due to goiter operations by different surgeons. The patients were totally out of connection with their surroundings, could not do any work and were in a desperate position in every way. The patient I am going to discuss now has a total paralysis from another cause—i. e., aberrant thyroid.

A short perusal of the literature showed that in the report of the following observation may be apropos. In February, 1926, a well developed woman, aged about 40, came in to my office with great difficulty in breathing, loud expiratory and inspiratory stridor. Her lips were blue, the suprasternal parts and the sides of the neck protruded like balloons at each expiration and sunk in deep in each inspiration. The difficulty started several years before and gradually became worse, so that she had not slept a night through for many months, the last six weeks sleeping only five to ten minutes at a time, sitting in a chair and resting her arms and head on the

back of a second chair. She had a harsh barking cough, and her voice had the same harsh monotonous sound. The laryngoscopic examination showed the upper larynx free, the vocal cords pinkish, in close apposition, actively immovable, passively closing valvelike at each in- and expiration. There was total paralysis of both vocal cords, of unknown cause. For treatment only intubation or tracheotomy could be considered. With the constant forceful coughing, the tube would be coughed out every few minutes. Tracheotomy was decided upon. The operation gave no information as to the etiology. She was kept in the hospital for several weeks for observation. X-ray examination, Wassermann tests, etc., were made, without result.

I did not see her for nearly two years, but on December 23, 1927, she again called me. She had a bad cold, was coughing incessantly, and I was struck by the unquestionably myxedematous expression of her face. It was difficult to ascertain the position or size of the thyroid, as the region was flabby, soft, moving in and out with each respiration, since it was implicated in the ballooning. After two days of thyroid medication her general condition improved and the myxedematous expression disappeared. There was never any difficulty in swallowing; an esophageal bougie No. 40 passed easily. X-ray examination of the chest showed bronchiectasis but no tuberculous foci. Then we took a transverse picture of the neck and recognized the following most peculiar findings: The course of the esophagus and the thyroid cartilage were very plain. On a level with the cricoid there was a swelling between the trachea and the esophagus 4 cm. long, protruding into the esophagus 1 cm. Another similar swelling between the vertebral column and the esophagus protruded 1 cm. into the lumen of the esophagus from behind, just above the anterior one. The lumen of the esophagus had a bayonet shaped course between the two swellings.

Malignancy could be excluded, since the swelling was soft, freely movable, changeable in size, and because, as the woman said, for years she was sometimes better, at other times worse.

A few years ago I presented before this Society another patient with an aberrant thyroid, a woman who developed increasing difficulty in breathing during the last weeks of pregnancy, at the same time that an otherwise hardly noticeable goiter grew enormously in size. A tumor from the rear left wall filled the whole cavum of the larynx, and left only the anterior right part free. Tracheotomy was performed, and two days later the woman was confined, which was rather difficult, as she could not press. A few days later, the tracheotomy tube was removed. With the help of a galvanocautery the tumor shrunk completely and the goiter diminished in size. The woman has since had another child without any difficulty in the throat.

**Fifth Year Otolaryngology in the Average General Hospital.**

DR. AUSTIN A. HAYDEN presented a paper entitled "Fifth Year Otolaryngology in the Average General Hospital."

(AUTHOR'S ABSTRACT.)

Otolaryngology in this paper was used as a vehicle for the conveyance of some thoughts regarding internships that had occurred to the essayist in his experience as attending otolaryngologist and chairman of the intern committee of a 200 bed general hospital of the "closed-open" type, and not closely attached to a medical college.

The fifth or hospital year is the most important of the entire medical course and deserves a constructive, comprehensive and well correlated plan of instruction and training. Otolaryngology should endeavor to interest the intern in medical organization. The hospital clinical conferences, which the fifth year student should be required to attend, will usually furnish his first actual opportunity to participate personally in a medical meeting. Attendance at all accessible medical meetings should be encouraged. The value of medical journals and the advisability of individual subscription to certain of these at the beginning of the internship cannot be too strongly emphasized as the best means of laying the foundation for a personal medical library. Interest should be stimulated in sound medical economics.

Comprehensive instruction and adequate training in otolaryngology are obviously impossible in the short time allotted to this specialty. The work should be so presented that those who wish to specialize will be inspired to enter the specialty of their own choice by the precept and example of their attending men, while the others will go into general practice with the feeling that the development of modern medicine has been so great that an individual brain cannot envisage the entirety of its scope, and no single pair of hands can master the endless and ever changing variety of its technical details.

The head of the department of otolaryngology must see that the hospital is provided with adequate examining, treatment and operating rooms, and a complete equipment of the instruments and apparatus necessary for efficient work. He should teach the intern, by his own example, the value of early and frequent consultation with other departments, as well as with other otolaryngologists. The intern should be impressed with the fact that he is quite apt to receive from his fifth year only what he himself puts into it, and that willing, cheerful service rendered to patients, attending men and hospital attaches seldom goes unrewarded. He must be taught always to compile his case histories carefully around the outstanding otolaryngologic symptoms of which the patient complains and properly correlate them with the observations of the general physical examination. Assistance at mastoid and other major operations

on the ear is all the operative work that should be permitted. Whether he shall perform myringotomies, even under proper supervision, is an open question.

In rhinology the local examination should be careful and detailed, and the periodicity and seasonability of symptoms should be studied. The application of casts, splints, posterior nasal packs and other appliances should be explained. The recognition of accessory nasal sinus disease must be taught. The advantage of photography, plaster casts, wax molds and roentgenograms should be emphasized. The treatment and, as far as possible, the prevention of head colds in infants and adults should be set forth in detail, together with the use of the local applications that give so much relief to the sufferer from the various forms of atrophic rhinitis with their accompanying laryngopharyngitis and chronic sinusitis. That the nasal cavity and its adjoining sinuses may lodge foreign bodies and new growths, and that congenital syphilis may here first betray itself should be emphasized. The operating room training of the intern in rhinology should be confined to observation and assistance.

In laryngology the history and physical examination should be thorough and painstaking. The conformation of the jaws and teeth and the general condition of the latter should be carefully observed. The appearance of the larynx, its contents and adnexa, as seen through the laryngeal mirror, and the condition of the nasopharynx should be ascertained when specially indicated, if not as a routine. Throat cultures before admission, diphtheria isolation and quarantine regulations should be explained. Intubation and emergency tracheotomy should be demonstrated, and more radical measures discussed and witnessed whenever possible.

The intern should be taught how to do tonsil and adenoid operations by the attending otolaryngologist, and allowed to do them under proper supervision if he shows average technical skill. If this work is not taught by the specialist the intern will get the work, less well, from general surgeons and practitioners who do so much of it in the general hospitals.

If otolaryngology blazes a trail in the new development of medical education which other specialties can adapt to their own particular needs, it will contribute to the betterment of the profession and the welfare of the public in general.

#### DISCUSSION.

DR. N. P. COLWELL (secretary, Council on Medical Education, American Medical Association), said he was glad that Dr. Hayden referred to the ages at graduation. Years ago, when only a high school education was required for admission, the average boy was higher than at present. The reason was that under the high school requirement pupils would wander around trying various callings before finally trying medicine, and some of these graduated at con-

siderably advanced ages. The average age of 26.8 is due to the large number of men who go on and get degrees before entering medical school. The percentage of graduates who also have baccalaureate degrees has increased since 1915 until now about 65 or 70 per cent of those who enter medicine enter with such degrees. The hospital internship situation has changed tremendously since 1912. In 1912, the Council on Education tried to find out how many hospitals would take interns. In some of the hospitals the interns merely had the duties of an orderly, pushing carts or bandaging, but doing little that would help them in the recognition and treatment of diseases. Later, however, and since the war particularly, the number of hospitals seeking interns has been so increased that many more are seeking interns than can get them, and that has made it possible to increase the standard of hospital work. During the last two years they have been able to put through a requirement which could not have been required even a few years ago. Now the hospital to be approved for intern training must hold autopsies on at least 10 per cent of deaths and the autopsies are to be taken up at staff conferences. That is developing in the hospitals a type of medical education which is worth something and shows whether the diagnoses are accurate. The comparison of antemortem and postmortem findings is of value both to intern and staff members. It also establishes a voluntary check over the members of the hospital staff. At one time in the medical schools the professors of medicine apparently did not care to have their diagnoses put to the test. Some of the younger teachers, however, in cooperation with some live wire pathologists, developed the clinicopathologic conferences and the students got the benefit.

The undergraduate schools are now giving so much better clinical experience to the students in their third and fourth years that the hospital training has to be correspondingly improved. The hospital internship now represents a rounding out process of the student's undergraduate education. It is the basis for further study leading toward a specialization. The question is still being discussed as to whether the rotating service furnishes the best training or whether a straight service is better, and whether the student should not go more directly into a specialty than with the rotating service. His power of diagnosis and his ability to send a patient to the proper specialist is more apt to be developed under a rotating service. The only hospitals in which the council has been willing to recognize the straight intern service are those connected with high grade medical schools. Until the time comes when the student's ability to make diagnoses is developed during the undergraduate period, there are objections to the straight service.

So far they have not taken up the curriculum to be followed by the student during his internship. Because of the short time the student spends in each service, unless that time is devoted largely to methods of diagnosis and indications for operation, the best part

of the student's training is lost. With the rotating internship as a basis, the means for further work toward a specialization has also advanced. Since 1912 better facilities have been developed for a real training in the specialties.

Five years ago a large special hospital in the East was visited which made use of interns immediately out of medical schools. One of the staff men remarked, "We have just had a meeting with the interns in the eye department this morning. They wanted more opportunity for performing operations. They said that the men on the ear service were obtaining more than those on the eye service." They were told that it could possibly be arranged. That hospital was allowing students coming directly from undergraduate schools to come there and perform operations. There was no doubt of the safety of the patients in the hospital because of the high grade men who were superintending the work. But how about the diagnoses of their own patients when they went into practice? In that hospital one of the best eye pathologists in the country and a professor at Harvard Medical School gave a splendid course in the anatomy of the eye, ear, nose and throat, and the better interns in that institution were voluntarily taking these courses. The staff was asked whether all the men should not be required to take these courses. Now all physicians who enter the hospital are required to have obtained a general intern training and also must take the courses in anatomy and pathology of the specialty. That hospital now affords the opportunity for an excellent training leading to specialization in eye and ear diseases. A similar development is happening in many hospitals throughout the country where thorough graduate work is being done. The question is, should the internship be a rotating service so as to thoroughly develop one's power of diagnosis, or should it be a straight service so as to direct the student immediately into a specialty? Just how much of each specialty should be given to interns in a general course, and how much should be left for a special course?

DR. M. T. MACEachern (Director, Hospital Activities, American College of Surgeons), stated that they want to standardize the arrangements and the surroundings the doctors and nurses work in so that they may have the best environment possible in all parts of the United States and Canada. They have a committee, of which Dr. Beck is chairman, and are hoping to bring out some valuable principles for the hospital trustees and administrators, and once they know they will help greatly in carrying it out. From a survey of 2,582 hospitals they find there are two things lacking: First, proper organization of the department. In by far the greater majority of the hospitals there may be very good operating facilities, but the patients are scattered through the institution, not always near treatment rooms, and certainly not always having the specialized nursing many require. They find there is need to teach hospital people that special patients con-

stitute a special group that needs grouping together in a proper department to facilitate proper treatment and nursing. Secondly, specialists are very busy and do not give the interns individual or collective teaching and instruction. There is lack of cooperation between interns and specialists on the staff, and that must be remedied. It is not the hospital trustees, but the surgeons themselves who do not like to see the segregation of the special branches of surgery. Regarding interns, Dr. Colwell has been the real mainspring for years behind this tremendous cleanup of the medical schools and the improvements in hospitals from the standpoint of medical education. Dr. MacEachern suggested three things he thought interns should have in their services, even if it is only six weeks. They should be taught to take careful histories of their patients. He did not think that histories in most hospitals on the eye, ear, nose and throat cases are as good as they should be. To collect clinical data which will help in research, all the histories should be complete. In addition to careful history taking, they should be taught diagnosis and the use of instruments in diagnosis. They should major in this in hospitals in the first year, particularly in this specialty. Many of these men will go into country practice and should know diagnosis so thoroughly that they will know when to call in the consultant. Further, he should know routine treatment. Young doctors are spurred on by giving them a taste of surgery in their intern year, and they go out with great bravery until they have had one or two reverses. He thought well to allow them to do little other than palliative treatment during their first year. The intern's chief work should be diagnosis. In specializing, his experience with five men who are now outstanding specialists, was as follows: He advised them to go to the country to earn sufficient money to study for two years in the best intensive clinics, after spending five years in general work. The men who spent the most time there became general men first, with grounding in pathology, X-ray and anesthesia, and are today very successful. If all young men would take a couple of years or three or four years in all branches, particularly pathology and laboratory work, and then spend a year or two in some industrial camp to make sufficient money to study for two or three years a specialty, he believed they would be successful.

DR. GEORGE E. SHAMBAUGH expressed his interest in listening to Dr. Hayden's talk and the discussion, and his full accord with the views expressed by Dr. Hayden and his conclusions regarding what we should try to do for interns in their general intern year. The question is exactly the one which the medical school has to decide in providing training in Otolaryngology for the undergraduate medical course, since the hospital year is only a continuation of this same work, which is that of preparing men for the practice of general medicine. A school providing undergraduate medical training does not aim to make specialists of its stu-

dents, and the question of what we should try to teach men in special fields during their hospital year is the same question which the medical college has endeavored to answer. He thought a decided change in the methods of instructing undergraduate students in Otolaryngology had taken place during the last twenty-five years. At Rush Medical College, for example, the old practice was to give the students their clinical instruction by means of the amphitheater clinic where operations on the nose and throat were the pabulum provided for the students. Now they aim to teach students how to make a proper examination of the nose and throat and ear, how to recognize the normal structures, how to use the special instruments for making these examinations, and something of how to make a diagnosis of the more common lesions. They may also give some attention to simpler local treatment, but the thing they do not do is to feed them up on operative work. Dr. Hayden brought up the question whether in the hospital year these men preparing for general practice should not be taught operations on the nose. That is what the student wants and one must ask whether they are unjust to the students. His answer to this question was very definite. He is interested in teaching operative work but not to individuals who have not had enough background in clinical experience to recognize when operations should be done. No practitioner of general medicine should be taught operative work in special fields. The country is flooded already with unnecessary operations, particularly in Otolaryngology, by general practitioners who have learned a smattering of technic of operative work, but have never had the opportunity of acquiring any proper appreciation of when these operations should be done. Some years ago an intern in the Presbyterian Hospital came to him with the request that he be allowed to go into the Central Free Dispensary and do three operations—a tonsil operation, a septum operation, and a turbinectomy. When he asked him why he wished to be taught how to do these operations, his reply was, "I am expecting to enter general practice in a certain city, and have been told by the doctors in this locality that it would be rather slow work for the first ten or fifteen years, and that I would have to expect to make a living doing tinkering operations on the nose and throat before I got a practice as an internist built up." He had a similar experience with a young man who had served as an intern under him in the Presbyterian Hospital, a number of years ago. That intern came to him one day and told him he had been having some very interesting work on the side, and related that he had made an arrangement with a physician who was taking care of some dispensary work by which he had been able to operate upon whatever cases he brought into the dispensary. He asked him what he had done and was told that he had done nineteen tonsillectomies, nineteen adenectomies, nineteen uvulectomies and nineteen turbinectomies.



When asked why he was partial to nineteen, his reply was that this was all the patients he had been able to find. He had done these operations on each one of these patients. When asked what reason he had for taking out the turbinal bodies, he was somewhat confused and his reply was something as follows: "Some of these patients had headaches, some had ear trouble, others had catarrh." Dr. Shambaugh told him that he had not mentioned any proper indication for a turbinectomy and that the needless removal of the turbinal bodies had brought upon each one of these patients a condition which would continue to be an annoyance throughout the remainder of their lives. He was convinced that we have no business trying to teach general practitioners the technic of operative work in a special field. This work should be reserved for those who are able to build up the background in clinical experience sufficient for evaluating the proper indications for operative work. It is a relatively simple matter to teach anyone the technic of operative work. It is a very difficult and painstaking job to teach men when to do these operations.

DR. GEORGE F. FISKE said that for fifty-one years he had heard discussions along this line, going back to the time when Bellevue had two years—now they have five years. How is it possible to be protected from the man who is not a specialist? After four or five years in medical school, he thought one must go and work with some man who is a specialist for two or three years more.

DR. JOSEPH C. BECK stated that up to this time probably forty-nine men and women have gone out and practiced otolaryngology from under his direction, and not a single one had ever been told that he could operate even when he was through with his special course; there was nothing promised to him about becoming an operator. It had been his fortune to see these young men and women placed with some of his friends, and they have turned out to be very good men and women. So he had a very definite feeling that Dr. Fiske was right, if one was interested in having good, safe specialists. In his time there were not the many opportunities offered as at the present to learn a specialty. This is a very large country and there are men that are in practice in districts where specialists are very few. It seemed to him in teaching men as interns one should try to teach them emergency treatment and emergency operations, and that was about all. When the emergency operation is done the patient may be sent elsewhere for better care. The fundamental necessities in the education of a specialist are not when he begins to learn his specialty but his undergraduate work, and if schools will devote more time to pathology and anatomy when the student comes up for internship and subsequently for study he will have a better knowledge. There is really no time for the undergraduate to get better education in the fundamentals of a specialty. In the hospitals, standardized hospitals particularly, what is required is outdoor departments. That is where the men

learn, where the men can be taught, and he believed that was the best thing to do for interns as well as for educating specialists.

DR. A. H. ANDREWS was interested in hearing Dr. Beck say he had been instrumental in making forty-nine specialists, but thought he himself could take credit for keeping a greater number of men out of the specialty. He had taught them what he could and sent them back to general practice with a better understanding of this specialty. That was what he thought was needed. A specialist is not made by study. Training of the eye to see and the hand to feel and the mind to know what the eye sees and the hand feels, and judgment to know how to act under certain circumstances—that only careful training will give, and one cannot get that in six years of medical school. He was interested in Dr. Shambaugh's report of the men who had done so many operations. He was once holding an examination in a class which he had not taught, and asked them to give the indications for the removal of tonsils. They wrote pages and pages, but one man gave an interesting answer. He wrote out the question, "Give the indications for removal of tonsils." Then he gave the answer, "The presence of a tonsil." While interested in the subject of otolaryngology, one must remember that the obstetrician, the gynecologist, the ophthalmologist had all the other specialists are all equally interested in their subject, and for each of them to carry out such a program as Dr. Hayden had indicated for otolaryngologists would require ten, fifteen or twenty years in the hospital getting ready to practice medicine. One must give them the indications, teach them to recognize cases and then let them go—and may the Lord have mercy on their patients.

DR. HARRY L. POLLOCK agreed with Dr. Hayden regarding the fifth year. Those who work in hospitals customarily know what interns demand. They state that they are going out to do the so-called simple operations, and in his judgment it is far better to teach them to do them properly, as they have taught some general practitioners who came to their place. It is far better to teach them something than nothing at all, and while one cannot make specialists of them in six weeks, one can teach them the technic. They let them operate under their supervision, because they are going to do it anyway. If one did as Dr. Andrews did, told them not to take it up at all, that would be still better, but 90 per cent of the tonsil work is done by pediatricians and general surgeons, and he thought it better to teach them to do it properly than to let them mutilate the patients. If one had a large enough service he could do a lot toward teaching them.





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